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ABSTRACTS FROM THE LITERATURE RELATING TO CONDITIONS IN THE TRACHEA AND BRONCHI.

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In reviewing the literature for late 1939 and early 1940 dealing with the history, anatomy, physiology, pathology, diagnosis and treatment of conditions relating to the trachea and bronchi, the wealth of material was such, and especially so in the English language, that but few other articles are included. War injuries of the trachea and bronchi are discussed.

In the historian's notebook, a column in the *Ohio State Medical Journal*, R. A. Aszling[1] gives the details of the removal of a foreign body from the airways. It is entitled, "An Early Tracheotomy," and states that Dr. J. C. Reeves, of Dayton, Ohio, removed a three-inch shawl pin from the lower trachea of a girl, age 8 years. Because of urgent symptoms of respiratory distress, Dr. Reeves proceeds with the operation, but fortifying his conscience with the dictum of Trousseau to "operate slowly, very slowly." Ice water was used to control the bleeding before opening the trachea; however, in opening the trachea some large veins were divided that poured blood into the air passages. Introducing his finger into the trachea, he passed a hook around the pin and withdrew the foreign body "by bending it out." Because of the blood in the airways, the child's breathing was so embarrassed that it had stopped. After clearing out the clots of blood, he closed the tracheal wounds with his fingers, and began mouth to mouth respiration. Shortly, she again began breathing. Dr. Reeves further states that "I felt considerable anxiety as to her

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recovery but within 48 hours she was out of danger, and her convalescence was uninterrupted." This occurred May 10, 1869.

The diagnosis was based on the history given by the mother, the child's statement that she had swallowed the pin, and on the physical signs of labored and embarrassed breathing.

The location of the foreign body was determined on the loss of expansion of the left chest, and much diminished breathing on inspiration over the same area. Paroxysmal coughing was a distressing symptom. "There could be no doubt that the pin was in the trachea, with its head in the left bronchus." Dr. Reeves proved this statement by removing the pin from this location.

Hulse and Curtis[2] report a tracheal accessory lung, and a description of the bronchoscopy is as follows:

"Instead of the usual two openings, denoting the origin of the right and left main stem bronchi, there were three. Each of these were approximately the same size." The Roentgenogram accompanying this report shows the condition very plainly.

Nicholson and Trimby[3] conclude that the rhythmic changes in the bronchial calibre synchronous with respirations are not reflex but probably purely passive. Finally, when one considers the characteristics of smooth muscle contraction and extreme variation in the respiratory frequency which may occur, it is very difficult to conceive of the bronchial muscle actively participating in each respiration. Certainly, no comparable example of such a synchronization of activity in smooth and skeletal muscles is available.

An article on the Embryology, Development and Growth of Structural Elements of the Larynx and Trachea, by Yokate,[4] will be of great interest if a proper translation were available. It is the only one appearing in the literature during the past year.

A very comprehensive Symposium on Peroral Endoscopy[5] appeared in THE LARYNGOSCOPE, 49:12,1160-1198. It contains articles dealing with the Inception and Development of Peroral Endoscopy, by M. C. Myerson; Anesthesia Methods, by Robert L. Moorhead; The Management of the Bron-

choscopic Clinic, by David H. Jones; Indications for Laryngoscopy and Bronchoscopy, by Rudolph Kramer; Indications for Esophagoscopy, Gastroscopy and Duodenoscopy, by Charles J. Imperatori; Contraindications to Endoscopy and Accidents, by John D. Kernan; Single and Biplane Fluoroscopy, by Frederick M. Law; and Pneumonograph, by George R. Brighton.

Martin[6] states that he feels that the bronchoscopist must give an initial and final diagnosis, particularly in malignant disease of the lung. He gives a very candid statement to one desiring to practice endoscopy in his country. One would gather from Mr. Martin's paper that the onus of not making a diagnosis of carcinoma of the lung is a lack of co-operation between the physician and the bronchoscopist.

It has been shown that in a recent study of 175 cases by Craver, and quoted in this review, that their experience did not bear out this assertion that bronchial carcinoma can be diagnosed early and with certainty in a very high proportion of cases. If aspiration biopsy had not been done, 36.5 per cent of cases would not have been diagnosed. Their reliance in diagnosing bronchiogenic carcinoma has been on aspiration biopsy, and within the past four years approximately 40 aspiration biopsies to 25 by bronchoscopy have been done.

The study of bronchology in Great Britain has been greatly hampered, as Mr. Martin so clearly states, by regulations and permits that interfere with practice on animals, and while cadaver bronchoscopy is practiced, the opportunity of examining living tissues must be confined to humans.

Free fascia grafts[7] have been successfully employed in dogs to repair defects in the trachea and bronchi, after local resection of full thickness segments of these viscera, as well as after lobectomy and total pneumonectomy. The 20 animals investigated all survived the operative procedure and remained entirely well throughout the duration of the experiment. The wounds healed per priman. There were no instances of subcutaneous emphysema, pneumothorax, hydrothorax or pleural empyema. There was in none of the animals any appreciable encroachment upon the calibre of the trachea or bronchus at the site of the application of the graft. Regeneration of the respiratory mucosa, already in evidence at the borders of the

defect, after one week was complete. At the end of the second week, the cells, at first cuboidal in shape and sparsely ciliated, quickly assumed the pseudostratified appearance and acquired their normal complement of cilia and goblet cells.

Its greatest field of usefulness appears at the moment to be in those instances of lobectomy or total pneumonectomy where it is necessary to transect the bronchus exactly at its point of bifurcation from the main bronchus or the trachea, respectively, and where no long bronchial stump is left that can be closed by the usual methods.

The cardinal signs[8] of obstructive emphysema on the skiagram are:

a. Greater transparency of the lung field on the affected side.

b. There may be displacement of the heart toward the free side.

c. It may be noted on screening that the displacement of the heart varies with respiration.

d. The dome of the diaphragm on the affected side may be depressed and flattened, and on screening its excursion may be found to be limited.

The signs of emphysema are most obvious at the end of expiration, and in all cases of doubt a film should be exposed at this phase.

Traumatic rupture[9] of the bronchi is compatible with life, providing the patient survives the immediate effect of injury; namely, shock, hemorrhage, pneumothorax and the possible development of suppuration.

The majority of benign bronchial tumors[10] fall in the category of benign adenoma. Pathologists still disagree on many important points, including the degree of malignancy. The history of a characteristic type of hemoptysis over a period of years, together with physical signs and bronchoscopic findings, should receive equal consideration with biopsy reports in diagnosing this type of growth. Atelectasis may afford a clue by pointing to the need of a diagnostic bronchoscopy. Artificial pneumothorax should be considered prior to bron-

choscopic removal or destruction by electrocoagulation because of its value in reducing the incidence of posthemorrhagic aspiration pneumonia. Treatment by bronchoscopic methods has resulted in many apparent cures or arrests and should be tried first. When this is unsuccessful, lobectomy and pneumonectomy offer an increasingly favorable outlook.

Carcinoma of the trachea[11] is rather of uncommon occurrence. Miller describes such a case of primary carcinoma of the trachea in a patient, age 65 years, diagnosed antemortum, with complete autopsy report.

The bronchomycoses,[12] while not rare, are not discovered with great frequency. The signs and symptoms are similar to tuberculosis and often at first thought to be tuberculous in origin. The final diagnosis rests on the discovery and identification of the offending organism. The Roentgen changes are suggestive of a differential diagnosis in some instances, but usually one can only suggest the presence of a fungus infection. It should be emphasized that the presence of fungi in the sputum does not in itself justify the diagnosis of a primary fungus infection, but it should indicate further studies to determine what rôle they play in the patient's problem.

Actinomycosis[13] monilia, oidium, blastomycosis, geotrichium, coccidioidal granuloma, torula and sporothrix were the organisms found. Many sputum examinations may be necessary. Lesions frequently resemble tuberculosis, and in some instances there is a studding along the bronchi. Monilia infections frequently assume this beading along the bronchi. There is no typical Roentgenographic appearance of these lesions in the lungs, and the diagnosis can seldom be made from the Roentgenogram alone.

Davison[14] concludes that in laryngotracheobronchitis direct laryngoscopy is essential for accurate diagnosis. Sulfanilamide is of therapeutic benefit when Str. hemolyticus is the infecting organism. Subglottic edema is less marked if the patient is kept in cool moist air, rather than in warm moist air. The use of atropine and of sedatives is contraindicated in the treatment of this disease. The development of pneumonia can usually be prevented if treatment is begun early and if the bronchi are at all times kept patent by removal of secretions and obstructing crusts. Bronchoscopic equipment

is essential for the management of patients with this disease. Tracheotomy rather than intubation is indicated when obstructive symptoms become marked. The mortality for this series of 17 cases was 11.7 per cent.

Diagnostic methods in bronchiogenic carcinoma by Craver[15] are based on 175 cases. He advocates: Bronchoscopy; thoracotomy; expectorated tissue examination; aspiration biopsy of lung; lymph nodes excused; aspiration of rib (direct invasion from lung); confirmed by autopsy.

The histologic types were: Squamous and epidermoid, 49 per cent; bronchiogenic, 9 per cent; adenocarcinoma, 11 per cent; anaplastic carcinoma, 11 per cent; "carcinoma," 21 per cent.

As a result of this study, it is believed that aspiration biopsy is essential for an early diagnosis of a large proportion of cases of carcinoma of the lung, and that treatment of this disease by high voltage Roentgen irradiation can accomplish a great deal in a palliative way, while possibilities of improved technique of irradiation promise considerably improved results, which in the majority of cases is not suitable for surgical intervention.

A histopathologic study of 40 necropsies is presented by Rabinovitch, Hochberg and Lederer.[16] A classification of these tumors is offered to include all the forms of primary malignant neoplasia which may be encountered in the bronchial tree. The unicellular theory seems to hold true in explaining the various forms of malignant growths that may arise in the bronchial tree.

Tuberculous tracheobronchitis[17] has an important bearing on the prognosis and treatment of pulmonary tuberculosis. It is suggested that more bronchoscopic examinations be done in sanatoria, not only to enrich our knowledge of tracheobronchial tuberculosis but also with the more important view of a more rational and better treatment of the patient. There is no accepted method of treatment of tracheobronchial lesions, but 30 per cent silver nitrate locally applied seems to have some value, particularly in assisting in the healing of early lesions.

Pierce and Curtzwiler[18] conclude that upon examination of serial chest films, tuberculous tracheobronchitis may be sug-

gested or indicated to the Roentgenologist by any one of the following:

a. Atelectasis.

b. Spontaneous intermittent atelectasis.

c. Extent of Roentgen pathology not compatible with clinical course.

d. Degree of collapse greater than expected from incident lesion.

e. Sudden increased atelectasis with collapse of therapy.

f. Visible change in the bronchi.

g. Oechsli sign (this is a thickening along the bronchovascular trunk leading from the parenchymal lesion to the root of the lung, with accented visibility of the bronchial lumen).

h. Tennis ball cavity (this is due to an obstruction of the bronchus leading to the cavity; this should be suspected if after satisfactory collapse therapy of the surrounding lung and particularly should the cavity seem to increase in size).

i. Lobular emphysema in a patient with positive sputum.

Summarizing a study of 400 cases of bronchiectasis, Perry and King[19] state that: 49 per cent developed the disease after an acute respiratory infection; 69 per cent started in the first or second decade (42 per cent in the first, and 27 per cent in the second). There is a strong impression that patients who develop bronchiectasis before the age of 10 years do not live beyond 40 years, and of the 59 patients who had reached the age of 40 years or over, 15 per cent had the onset of the disease in the first decade. The disease rarely spreads beyond the lobe or lobes originally involved. A 12-year study of the entire group showed the mortality in the nonsurgically treated cases 26 per cent, 41 per cent of these dying within five years of the onset, and 15 per cent living 20 years or longer after the onset. The best risks were undoubtedly selected for surgery, but a review of the nonsurgical group showed that 50 per cent had unilateral disease, and that 75 per cent were anatomically suitable for operation. The operation mortality in 122 modern type lobectomies performed on 116 patients was approximately 3 per cent.

The surgical problems of the war and personal experience by Lockwood[20] are of great interest. Casualties caused by close-up shell or bomb explosion cause death without an apparent wound. These casualties have a rupture of the visceral pleura or a complete collapse of the lung due to suction of air incidental to the blast, or due to reflex or bends. Immediately on finding such a case, it is advised "to place a handkerchief or piece of gauze over the mouth, hold the nose and blow hard into the lungs." The lungs must be kept inflated and it may be necessary to aspirate the pleural cavity, or even leave a small needle in the chest wall until a positive pressure within the thorax no longer occurs.

Laurens, Aubry and Lemariey[21] describe injuries of the larynx and trachea following gunshot injuries, and discuss the functional and traumatic neuroses, also the incidence of infection resulting in stenosis of the airway. Trauma of the trachea should be followed in most instances by prompt tracheotomy. Intubation is rarely to be preferred to tracheotomy in these injuries.

Injuries to the trachea[22] require emergency treatment and it must be borne in mind that not only the immediate emergency be met but the future possibilities must be considered. These injuries are comparatively infrequent when compared to the more exposed skull. Protection is provided by the projecting lower jaw, which is lowered in threatening danger, still further narrowing the distance between it and the sternum. Crushing (compression and squeezing) may result from stray shots, etc. That fracture of the larynx or trachea does not occur is likely due to the elasticity of the youthful larynx and trachea. Following these injuries, the pathological change consists in varying amounts of submucous hemorrhages in the subcutaneous cellular tissue and muscles, also edema and occasional emphysema, and, as a sequel to these injuries, a perichondritis, depending on the degree of injury. In these injuries depending on the amount of dyspnea, cyanosis, emphysema, shock, will depend the treatment. Tracheotomy should be done immediately, and the patient sent further back along the lines of communication. Bullet wounds of the neck frequently pierce the trachea and the soldier dies before help can reach him on the battlefield. For those who reach the hospital, the outlook is not so bad.

The physical standards[23] governing the physical examination of registrants by the examining physician of the Selective Service, among many disqualifications in the mouth, nose, fauces, trachea, esophagus and larynx examinations, a permanent tracheotomy is considered a permanent defect and therefore a disqualification.

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A REVIEW OF RECONSTRUCTIVE SURGERY OF THE FACE.*

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NOSE.

Interest in the correction of nasal deformities has been stimulated by the recent use of preserved cartilage transplants. O'Connor[62] and Pierce[63] collect such cartilage under sterile or unsterile conditions, strip it of perichondrium and store it in 1:1,000 aqueous merthiolate in a refrigerator. The incidence of infections in their transplants has been low, but all of them have occurred in noses. Because of this they advocate making intranasal or columellar drainage wounds and irrigating the graft bed with a merthiolate-saline solution. Partial or complete absorption of the transplants in their series was only an occasional finding and they believe that the preserved cartilage curls less than fresh. Brown[8] states, however, that absorption takes place to some extent in all instances and complete loss may occur by a gradual process without visible reaction over a period of several months. This process is not always uniform and the transplant may appear actually scalloped. He points out that the experimental implantation of pieces in animals or in the abdominal wall of patients is not comparable to the clinical use because these beds are better than those about the nose and face, where the material is usually needed. He finds that the preserved implants do not withstand infection well, but they have healed in satisfactorily following drainage from the wound in some instances. Childrey[20] advises placing sulfathiazol in the wounds with the transplants and giving it orally to prevent infections. Peer[69] obtained microscopic sections of six pieces of preserved cartilage which had been implanted from nine and one-half months to two years and found that all of them showed progressive invasion by fibrous tissue and partial absorption. Eight segments of autocartilage studied simi-

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larly showed no invasion nor absorption. Two autografts buried four and one-half years and six years appeared as living cartilage. After having used preserved cartilage since 1928, Brown's conclusion is that it should be used only as a second choice substitution when, for some reason, fresh cartilage is not advisable (see Figs. 1 and 2).

Wolfe[83] prefers ivory to cartilage, stating that the latter often produces an abnormal midline ridge due to absorption. Cohen[24] prefers a mixed cantilever type of graft, using carti-

Fig. 1. Diagram of L-shaped nasal cartilage transplant. (a) Above. Side view of single piece of cartilage to restore nasal contour with support at the nasal spine and above. This type can be put in luetic noses with only a small amount of tissue left in the columella. If the whole cartilage is lost but the mucosa remains, even a larger sheet may be used practically to replace the septum. The upper end is always placed under the periosteum if possible, and this applies to all regions; if only a small pocket of periosteum can be prepared, the best possible support is then obtained. The large piece is best obtained by cutting it out of an angle or where two cartilages are united. (b) Top view, showing breadth above and thinning below to avoid widening of the tip and columella. If necessary to gain lateral bulk, wide side wings may be left on or extra pieces put in. Both views are diagrammatic. The general shape is cut out and then trimmed down to pattern.

lage for the septal and columellar portion, and bone for the dorsum. Brown[8] prefers an L-shaped piece of cartilage which is cut in one piece, obtaining its support below at the nasal spine, above at the glabella, and along the septum if any is present. Young[84] advocates a cartilage graft shaped like a tent; the columellar strut is cut from across the end and left attached by a hinge of perichondrium, and similar lateral struts are turned down from either side. Harbert[39] uses ear cartilage as a simple dorsal implant, leaving the anterior perichondrium in the ear to regenerate. Warping is said to be less than in that obtained from a rib. Sarnoff[74] illustrates a case with a depression between the bony nose and the tip, follow-

ing a submucous resection. This was filled by rotating the upper lateral cartilages into the defect, after narrowing the bones. Cinelli[21] illustrates two luetic saddle noses which were repaired by a very extensive operation through a combination of intraoral, intranasal and external skin incisions, cutting the columella loose from the lip and inserting a two-piece rib graft.

Fig. 2. (a) Left. Deformity following autogenous cartilage transplant (done elsewhere) with calcification and distortion of the cartilage, rendering it useless. (b) Result of preserved cartilage transplant. Complete mobilization of the lateral bony walls was necessary for narrowing after the distorted cartilage was removed; through and through wires were put in for fixation. As it is often best not to combine an extensive osteoplastic with a fresh cartilage transplant, a preserved cartilage was used as a temporary subcutaneous prosthesis. It was healed promptly, however, and is in good condition more than two years later.

A. M. Brown[7] describes the "artistic anatomy" of the nose, noting that the congenital humped nose presents a hypertrophy of the whole nose, including the septum and external cartilages, while the traumatic nose usually presents only humping and widening of the bony portion.

Maliniac[53] discusses the prevention and treatment of late sequelae in corrective rhinoplasty. Though Joseph dressed his rhinoplasties with only a little adhesive and gauze, Aufricht[1] found that occasional hematomas followed this, and he has been using a pressure dressing for many years. The details of his method of using dental molding compound for this purpose are well described and he stresses the necessity

of evacuating all blood, sawdust and cartilage debris from the interstices of the nose before applying any splint. Pearlman[68] introduces a short metal nasal splint with slits cut on each side for easier bending, a combination chisel and periosteal elevator, and a four-bladed thumb forceps for introducing cartilage transplants.

Kopp[50] states that all surgical procedures for the relief of atrophic rhinitis depend upon decreasing the width of the nasal chambers. He thinks that this disease does not contraindicate an osteoplastic operation and is often helped by it.

Ivanissevich and Ferrari[41] illustrate the "Argentine method" of replacing the tip of the nose with a jump flap of the full thickness of the ear, using the thumb as a vehicle. Straith[76] uses an ordinary tubed vertical neck flap for this purpose but transposes the upper end as the skin from behind the ear more nearly matches that of the nasal tip.

Brown[13] has written a rather extensive review of the procedures used by him in the correction of various types of nasal deformity. Included are operations for reduction in size, deviations of the nose, fractures, lacerations of the soft parts, saddle nose, replacement of the skin covering, total and partial nasal restorations, retrusion, nasal deformities associated with cleft lip, rhinophyma, and the treatment of hemangiomas about the nose.

CLEFT PALATE AND LIP.

For complete single clefts, Ivy[42] prefers to close the anterior palate by a first stage Veau operation at the time of the lip closure, usually between the ages of 6 weeks and 3 months. The posterior portion is closed by the method of Axhausen not before the age of 2½-3 years. At this operation the lateral flaps are raised as for a von Langenbeck procedure but both major palatine arteries are cut and tied. The cleft edges of the palate are divided by splitting and not paring, and the nasal mucosa is elevated from the bone in such a manner that its continuity back over the soft palate is maintained. The palate is then closed in three layers: nasal mucosa, muscle and oral mucosa. Median clefts of the soft palate are likewise closed by the Axhausen procedure. For complete double clefts, he does a first stage Veau operation on one side, clos-

ing that side of the lip at the same time. The other side of the lip and palate are done a few weeks later and the remainder of the cleft is closed at 2½-3 years by the same Axhausen technique. For velopharyngeal insufficiency he advocates Dorrance's push-back or Brown's elongation operation.

Kazanjian[43] also prefers a two-stage operation for complete single palate clefts, closing the anterior portion first and the soft palate a few months later. Smith and Johnson[75] prefer Dorrance's method of closure for ordinary clefts and his push-back procedure for congenitally short palates, with some modifications of suture material and technique.

Brenizer[5, 6] uses wide bands of transplanted fascia placed across the nasal surface to take the tension off the suture line and to act as a scaffolding and support for the palate. He is unable to visualize the bands after operation but believes that they persist. Fascial or catgut bands are also utilized in closure of the lip, especially in double clefts. He sections the neck of the premaxilla and slides it back "like a drawer," after which the bands are sutured underneath the mucoperiosteum of the premaxilla and the alveolus on either side.

Brown[9] has found it possible to do double elongations of partially cleft palates to gain maximum length and at the same time preserve the levator and sphincter actions. The procedure is used in palates in which there is so little tissue that one elongation will not give sufficient length for total nasopharyngeal closure. His regular elongation with preservation of the arteries is done at the first stage. At the second operation, the originally elongated portion is split from before backward and from side to side, care being taken not to break through into the nose. The arteries are sacrificed at this time and a second elongation done. The cleft may be closed at either stage as desired. As a corollary to this procedure, he also found that elongations could be done in totally cleft palates which have been satisfactorily closed and in which additional length is desirable. He emphasizes that for best speech the palate must not only be of sufficient length, but the velum must be soft and pliable and its motor mechanism intact. In any procedure in which the posterior pillars are sutured together behind the uvula, or in which a posterior pharyngeal flap is sutured to the velum, a tight scar band is placed in the region where the most movement is essential.

For partial palate clefts, Kilner[48] does a Wardill pharyngoplasty followed by a V-Y procedure behind the incisor teeth (incision of Ganzer) to lengthen the palate. For total single and double clefts of the lip and palate he uses modifications of the Veau procedures.

In primary lip repairs, McIndoe[57] completely mobilizes the alar cartilage on the cleft side to correct the nostril deformity. Incisions are made on the inside above and below the cartilage and it is freed from the skin as a "bucket handle flap." This allows the cartilage to spring forward and take its proper place in the new skin mold. In secondary repairs, the same mobilization is followed by any necessary trimming and suture fixation to properly shape the nostril. Humby[40] fills out the depression of the nasal tip on the cleft side by swinging a flap of the normal alar cartilage over in front of the dome of the cartilage on the cleft side. Sometimes the major portion of the horizontal crus of the normal cartilage is used in the flap, but he has not found the collapse of the tip on the sound side to be very noticeable. Owens[64] reviews the history of operations for cleft lip and palate, stating that he prefers a modified Mirault procedure for single lips.

PROTRUDING EARS.

Davis and Kitlowski[29] call attention to the lack of a visible antihelix in many prominent ears and believe that this ridge should be reconstructed as the ear is set back. If the ear is held back against the scalp, the antihelix becomes visible and it is marked by green puncture marks along the ridge extending completely through the ear and into the postaural skin of the scalp. An ellipse of skin is thus demarcated, half on the back of the ear and half on the scalp, and excised. The cartilage is incised along the line of these dots and a narrow vertical ellipse taken out. The remaining proximal and distal pieces of cartilage are then sutured together back-to-back with their raw edges pointing forward to form the ridge of the new antihelix and the skin is closed. New and Erich[59] do a similar operation but use mattress sutures put in on the anterior surface of the ear to hold the new antihelix forward. The amount of tension on these sutures controls the prominence of the new ridge. MacCollum[51] outlines three procedures for the correction of these ear deformities. His second

method is essentially the above operation, except no skin is excised. In his first method, an ellipse of skin is excised and the cartilage is incised along the puncture marks. A narrow crescent of cartilage is then excised from the outer or distal portion and the cartilages are repositioned with the cut edge of the proximal piece placed anterior to the outer cartilage. Thus the cut edge of the proximal cartilage alone forms the new antihelix. In the third method, a narrower ellipse of skin is removed and a pear-shaped portion of cartilage is excised, the widest portion being at the base of the concha. The cartilages are then approximated back-to-back. In any operation in which skin is excised, the cartilages are sutured back to the galea before skin closure.

Rigg and Waldapfel[72] have devised a procedure for the correction of drooping or "dog" ear, a condition in which the upper portion of the ear is turned down and outward. An ellipse of skin is excised from the back of the upper ear and adjacent scalp. A rectangular flap of ear cartilage is then turned backward and inserted beneath the periosteum of the adjacent skull and the skin is closed.

RECONSTRUCTION OF EARS.

Padgett[66] illustrates some total reconstructions, using rib cartilage for the framework, a flap for the back and helix, and a free graft for the raw scalp surface. He uses a fairly wide vertical neck flap with the distal portion split into a small tubed flap for the helix and a larger flat portion for the back of the new ear. He prefers to wait until the individual has attained his full growth before operating, as the growth potential of a reconstructed ear is not certain. Attempts to reconstruct the canal are not thought practical. Kirkham[49] introduces an interesting variation, using a cadaver ear cartilage as a framework. As the cadaver ear cartilage behaves somewhat as a foreign body and may become loose from the surrounding structures, he makes holes through the transplant so that granulations may penetrate and fix its position. O'Connor and Pierce[63] use preserved rib cartilage for the framework.

FACIAL PARALYSIS.

Cleveland[23] reviews the rather voluminous literature between 1932 and 1938 on the treatment of facial paralysis.

Direct nerve suture and free nerve transplants, as introduced by Ballance and Duel, are established procedures when applicable. The trend seems to be away from preparing the grafts by allowing Wallerian degeneration to occur. Bentley (quoted by Tickle[77]) found that an equal amount of time was required for the return of function with degenerated and fresh grafts. He believes that very few neurones grow down through the tubules of the graft, most of them passing along the outside of the sheath. Bunnell and Boyes,[19] working on cats, also found that prepared grafts had no advantage over fresh ones. Sections showed some neurones growing through the graft, but most of them near the periphery. They explain this on a nutritional basis and advise using cable grafts of multiple small strands to permit better contact of most of the fibres with the surrounding lymph.

Interest in hypoglossal-facial anastomoses was somewhat renewed by an article published by Coleman.[25] Two excellent results are illustrated and the indications for and details of the operation are described. He has been impressed with the failure of the nerve-grafting and suture operations to restore emotional expression or to prevent mass movements of the face, and notes that it is impossible to correctly align the proximal and distal ends of the fibres supplying the individual muscle groups; however, he uses the anastomosis chiefly for cases in which there has been intracranial damage to the nerve. The hypoglossal nerve is usually preferred, but in loquacious patients it may be better to use the spinal accessory. If there is any residual paralysis or incomplete recovery of important muscle groups, Brown's operation[10] may be used as a supplementary procedure.

For those cases in which nerve anastomosis, suture or transplant is not applicable, various plastic procedures have been devised. Sheehan, Halle and others[23] have attached muscle and muscle-nerve pedicle flaps from the temporal and masseter muscles to the corner of the mouth. Sheehan has noted that the bulk of the flap is sometimes disfiguring, and makes a groove in the zygoma to accommodate it when this is the case. Brunner[18] uses a masseter muscle flap, doing all of the operating through the inside of the mouth. He believes that filaments of the Vth nerve grow out of the masseter flap and innervate the surrounding muscles of the face.

Brown[10] obtains balance and movement of the paralyzed face by means of transplanted fascial loops connecting the lower face with the most mobile portion of the temporalis muscle. Long strips of autogenous fascia are introduced

Fig. 3. (A) Complete facial paralysis—congenital. (B) Result of the described operation showing motor power of the (Vth nerve) temporal muscle elevating the lip in a slight smile to give some degree of emotional expression. (C, D) Later photographs to show persistence of result and also improvement in control of the face and in emotional expression.

through an incision in the hair-bearing temporal region. One strip is passed subcutaneously down to the philtrum or the middle of the upper lip and then back at a slightly lower level to form a loop. Another similar loop is passed down to the

corner of the mouth and the lateral portion of the lower lip. Both loops are then anchored to the tendon of the temporalis muscle just above the coronoid and any excess skin is excised before the temporal wound is closed. After any swelling and soreness has subsided, the conscious act of setting the closing muscles of the jaw produces some degree of emotional expres-

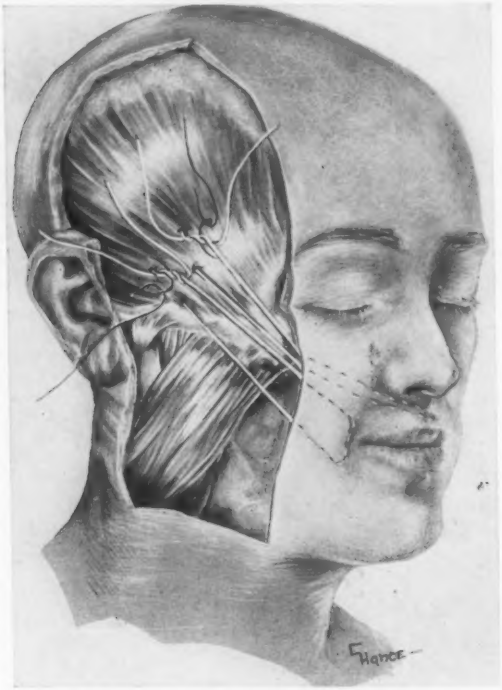

Fig. 4. Diagram of the free fascial loops' course through the face and fixation in the temporal muscle.

sion in that side of the face, including a slight smile and the production of a nasolabial fold (see Figs. 3, 4 and 5). The patient is trained in limiting the degree of movement on the sound side and in coordinating the movements of the two sides. After a preliminary period, this should become an

involuntary or subconscious function. Some improvement in any sagging of the lower lid is obtained by the above procedure. If the lid needs further support, this may be obtained by passing another strip of fascia from the temporal region through the lid to the opposite frontalis. No attempt is made to obtain Vth nerve motion for the lid as this would be too gross and too conscious. For any apparent exophthalmos present, the palpebral fissure can be narrowed by a small external canthoplasty.

Fig. 5. (A) Complete ptosis of the upper lid from a IIIrd nerve lesion, and paralysis of the frontalis (VIIth nerve) muscle from the local scar. (B and C) Elevation and some degree of emotional expression in the lid by attachment of lid to the forehead with fascia, and by a second loop of fascia from the temporal fascia through the tarsus to the opposite functioning frontalis.

EYE.

So many substances have been advocated to replace conjunctival loss that it is evident that all of them leave much to be desired. Clay and Baird[22] have been using grafts from the prepuce and labia minora and illustrate two such cases. These grafts have the appearance of conjunctiva and do not desquamate. Fetal membranes are used for this purpose by de Rötth.[31] The membranes were obtained by Caesarean section and implanted from one to 15 hours later. The transplants were used in six cases for symblepharon and for lining a socket in two instances. The chorionic surface is placed next to the wound, with amnion forming the free surface. The grafts took in each instance and for three weeks they showed no change. In the next weeks the grafts shrunk and 10 weeks after operation they seemed to have disappeared, but there was always more fornix left than before operation. Blair and Byars[4] use free skin grafts. The scar is dissected

out and the graft is inserted over a wet cotton or paraffin mold and the lids are closed with sutures. Some corneal irritation, and even transient ulceration, has been encountered, but this is believed due to technical errors rather than to any fault of the method.

For paralysis of the lower lid resulting from interruption of the nerve supply, Blair and Byars[4] have used autogenous fascial strips. Sometimes the strip is anchored on either side above in the frontalis muscle, being put through the lid as a hammock. In other cases a circumferential strip has been used through both lids and anchored above the eyebrow or in the temporal region. These have been combined with canthoplasties when indicated to narrow the palpebral fissure. For paralysis of the lower lid resulting from loss of substance or scarring in the neighborhood, a small forehead skin flap has been used as a hammock. Rosenburg[73] describes the treatment of ptosis by means of a fascial sling passed through the upper lid and anchored in the frontalis, combined with an elliptical excision of skin from the upper lid. The treatment of ptosis and of the eyelid deformities associated with facial paralysis was also included in Brown's[10] paper on the latter subject (see Fig. 5).

FACIAL REPAIRS.

Kazanjian and Converse[46] prefer osteoperiosteal grafts from the tibia for repair of defects of the frontal bone. The grafts are chiseled out in long strips and multiple fractures are made in them if necessary to get the proper curvature. They prefer an incision above or below the eyebrow if possible, as a hairless brow scar is often quite evident. New and Dix[58] use autogenous rib cartilage routinely for this purpose. Often it is necessary to suture several pieces together with fine silk to fill a large defect. At times the transplants are fixed to the frontal bone with small pieces of stainless steel surgical needles used as tacks. For small defects, overcorrection with a pad of fascia lata may suffice. They have tried free fat transplants but are not enthusiastic about them. Peer[70] also prefers autogenous cartilage, but has used preserved in some instances when the former was not available in sufficient quantity. Dermal grafts are used for shallow depressions.

Figi[34] has outlined some of the methods useful for the repair of defects resulting from the removal of extensive antral

tumors. Cheek perforations are closed with forehead or chest flaps, while any bony loss about the orbital border is repaired with cartilage or bone transplants. Any elevation of the upper lip is corrected by freeing the scarred attachment of the lip from the maxilla and skin grafting the inner surface. Displacement of the meatus of the parotid duct in one instance resulted in drainage of saliva into the nose and this required transplantation of the duct to the mouth.

New and Erich[60] have published a very clear and complete description of the various local flaps and transpositions that may be used for the closure of small lip defects. The repair of large defects with forehead flaps, tubed neck flaps or flaps from a distance is similarly well illustrated. Daland[26] excises large lip lesions in a rectangular manner and closes the defect by sliding lateral flaps in together. Wide undermining is necessary, together with lateral cuts extending from both lower angles of the rectangle. The orbicularis is separated from the other facial muscles at the angle of the mouth to permit satisfactory rotation of the upper lip down into the lower lip on each side. The procedure results in a rather small mouth opening in some instances.

FACIAL INJURIES.

The importance of complete diagnosis and early treatment is emphasized by most recent writers and is the subject of a paper by Foster.[35] Nearly everyone agrees that all dirt and oil should be scrubbed out of such wounds, perfect hemostasis obtained, and meticulous closure of the soft parts done. Brown and McDowell[11, 17] believe that replacement of any fractured bones at the time of primary repair is a keynote and describe the common fractures and their treatment. Conservative debridement is advocated so that parts of features will not be needlessly sacrificed. Whitham[82] illustrates fractures of the upper jaw, nose and zygoma, together with methods of late restoration with flaps and transplants. Bellinger[3] describes the treatment of jaw fractures with interdental wiring and with elastic traction, stating that instead of draining all of them which are compounded into the mouth, he prefers to wait and drain only those in which abscesses occur. E. D. D. Davis[27] writes that "a fracture of the mandible will not unite if the teeth on each side of the line of fracture are not

extracted." Brown[12] places a small dependent drain up to all fractures which are compounded into a tooth socket and does not extract the tooth in the fracture line early even if it is somewhat loose. This is believed to be of especial importance in molar fractures in which the tooth may prevent the tendency to upward displacement of the posterior fragment. Von Wedel[79] gives a good description of the care of the bones and soft parts in facial injuries.

MANDIBULAR OPERATIONS.

For replacement of resected portions of the jaw, Kazanjian[44] uses osteoperiosteal grafts from the tibia. The jaw should be immobilized in occlusion from the time of the resection until the transplant is done. In dealing with tumors of the ramus, the head and neck of the condyle should be left in if possible.

For retrusion of the jaw, he extracts the second molar tooth on each side and does an intraoral L-shaped osteotomy of the body in that region and slides it forward. The jaw is then held forward with interdental wires and an external appliance anchored to the forehead and upper incisor teeth. This procedure may be supplemented later by a bone or cartilage transplant in front of the chin. For prognathism, he does a horizontal section of the ramus and slides the jaw back unless it is associated with an open bite, in which case he does an oblique osteotomy in the body near the angle.

New and Erich[61] discuss the retruded chin, including etiological processes, varieties and the types of treatment. The corrective procedures, in general, follow six plans: *1.* various types of osteotomy through the horizontal or ascending rami to lengthen or advance the mandible; *2.* osteotomy through the horizontal rami associated with the use of bone grafts to lengthen the mandible; *3.* cartilage implants inserted behind the head of the condyles for advancement of the mandible; *4.* cartilage, bone, ivory and celluloid implants for building up the chin to normal contour; *5.* prosthetic appliances requiring the use of intraoral skin grafts for restoration of normal chin contour; and *6.* orthodontic methods. They point out that in those cases in which the chin has been built out with cartilage transplants, the lower lip may remain retruded.

This latter deformity is corrected by incising the fornix between the lip and alveolus, widening the sulcus and skin-grafting the resultant raw area. The lip is then maintained in its new forward position by wearing a prosthesis in the sulcus. Weisengreen[81] also reviews the operations for micrognathia and macrognathia, together with the methods of direct fixation of the fractured mandible.

For temporomandibular ankylosis, Kazanjian[45] removes a complete section of bone from the bottom of the sigmoid notch backward and downward. A sufficient amount of fascia to obliterate the dead space is then inserted between the ends. Giangrasso[36] does a linear osteotomy at the base of the neck of the condyle and interposes a piece of rubber glove between the fragments.

MISCELLANEOUS.

Mason, *et al.*,[55] illustrate a case of congenital lip pits in which there were two large oval sulci, one placed horizontally on either side of the midline. It is noted that during the late prenatal and early postnatal life, the glabrous and villous portions of the lip are ordinarily separated by a sulcus. These pits apparently represented persistence and deepening of portions of this sulcus; *i.e.*, a tendency to reversion to a double lower lip. They were treated by oval excision and advancing mucosa up out of the fornix to suture to the skin. Baxter[2] describes some similar lesions which he calls "congenital fistulas" of the lower lip. He has found that they may be too small to admit a fine probe, or may be as much as 0.5 cm. in diameter, and not infrequently mucous glands open directly into them. He advocates destroying the smaller ones by electrocoagulation and excising them if large.

Brown and Byars[14] report four- and seven-year cures of malignant melanomas which had metastasized to lymph glands. They believe that these lesions should not be regarded as entirely hopeless but should be treated by complete local excision and radical excision of the regional nodes. It was observed that many of the fatal lesions had been previously subjected to multiple electrodesiccations. They prefer to remove moles by cutting them out with the actual cautery, going down into the subcutaneous fat. In deciding which moles to remove, it is believed that all which are subjected

to chronic irritation, or which show evidence of growth, increase in vascularity, change of color, repeated infection or ulceration should be excised. The smooth, coal-black nevi should likewise be removed. Maliniac[52] treats small nevi by desiccation or by trichloracetic acid, except the flat ones, which are usually excised. For the larger ones, he prefers single- or multiple-stage excision, followed by rotating in adjacent skin flaps, or free skin grafting.

Brown and Byars[15] note that hemangiomata may be found in all degrees of severity from small, bluish-red blotches and port wine stains to large, venous engorgements and areas filled with bright red arterial blood. The blotches and deeper stains and even the large venous channels are often congenital and might be considered as local anomalies rather than tumors, but the bright red arterial type is apt to exhibit definite new growth characteristics. In infants and children, consideration of the "strawberry" or arterial type is most important because of their tendency to spread, and when present on the face to destroy features. Surgical excision or cautery destruction is the best treatment when their size and location are such that the treatment will not deform features or other contraindications are not present. Surface radiation may be used when the involvement is not deep and where applications can be made satisfactorily. In bulky lesions, where excision is not applicable, interstitial implantation of gold radon seeds is the method of choice. Ward and Covington[80] advocate surface radiation for most of the capillary and cavernous types. In the cavernous types, it is usually necessary to follow this up with interstitial electrocoagulation. It is noted that irradiation gives poor results in port wine stains. Solid carbon dioxide is used by them for very small lesions, residual patches, and areas about the eyelids and scrotum.

Gillies[37] describes the technique and uses of his well known tubed pedicle flap, illustrating the most common donor and recipient sites. He utilizes them for a wide variety of repairs, recommending them especially for restoration of the avulsed scalp, massive face reconstruction, large cheek defects, or when restoration of contour is necessary, total loss of lips and chin, the contracted neck, or the nose if a forehead flap is not used. Maltz[54] has experienced some difficulty in get-

ting the flaps to lie flat after untubing and therefore prefers to tube them in a reverse manner, with the raw surface on the outside. "The interrupted sutures of silk forming the tube can be tied in such manner as to prevent union of opposing edges of the wound. In such instances, the stitches eventually pull through and the tube unrolls by itself by the time it has to be transplanted."

In England, Rank[71] has been using Thiersch skin grafts to prevent contractures in burns. The percentage of loss due to infection is perhaps greater than that ordinarily seen in this country, but the results are well worth while. Padgett[65] has introduced a new instrument for cutting partial thickness skin grafts. It consists of a metal drum with an attached sliding knife blade which can be set at various distances apart. The drum is made adherent to the skin with rubber cement, thus forming a skin diaphragm for the knife to cut on. The drum surface measures 8 x 4 inches, so a split graft of 32 square inches or smaller can be obtained with it.

J. Staige Davis[28] writes of the value of small deep grafts for repairing surface defects and states that it has been possible to transfer them successfully after refrigeration for as long as five weeks. He prefers the anterior surface of the thigh for a donor area as the skin is thin and of about the correct tension. He believes the appearance to be less satisfactory than that of split grafts, but they are usually used in unexposed areas. The small deep grafts are thought to take in many instances on surfaces on which no larger graft could.

Uihlein[78] states that the cutis or dermal graft is being used increasingly, both in the repair of hernias and for restoring contour in plastic work. Eitel, using the Warburg technique, demonstrated that the dermis is more active and lives longer than fascia. Dean[30] describes this graft under the name "dermigraft" and apparently has used it for surface covering. A Thiersch graft is cut from the thigh and left attached at one end "to retain the blood supply intact," after which the dermal graft is removed and transplanted. The Thiersch graft is then sutured back down in place.

McCarroll[56] has noted that sensation returns simultaneously throughout free skin grafts, averaging 45 days in split and

60 days in full thickness grafts. In flaps, however, the sensation returns first near the pedicle, then near the area first attached, returning throughout on an average of 285 days.

Several interesting treatments of avulsed skin flaps have been recently proposed. Farmer[33] and Keyes[47] advocate careful cleansing, removal of all fat from the back, and replacement as a free full thickness graft. The "take" has not been perfect in the recorded cases but has been of a sufficient amount to make the procedure worth while in selected cases. Douglas[32] cuts pinch grafts from the avulsed skin flap and implants them.

The details of preoperative and postoperative care in reconstructive surgery are discussed by Brown, *et al.*[16] Among other things, the use of pressure dressings on all grafts, flaps, transplants and also on most ordinary wounds is recommended. It is believed that this minimizes the occurrence of hematomas and edema and, thereby, infection. The external bandage produces the pressure, but it can be transmitted to the wound by various materials. Marine sponges have been used for some time but have recently been largely supplanted by soft cotton mechanic's waste. This material is readily obtainable, inexpensive and packs nicely down into small crevices and folds to transmit an even pressure to an uneven surface.

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Metropolitan Building.

SYMPOSIUM ON BRAIN ABSCESS.

(a) – LATE COMPLICATIONS OF BRAIN ABSCESS.*

DR. IRA COHEN, New York.

To illustrate some of the late complications following brain abscess, I shall show four patients this evening. It is because these patients are to be presented that the subject of late complications appears first on the program and not in its chronological order.

When, for one reason or another, a brain abscess occurs, and when it is properly diagnosed; when an operation is performed and the patient is discharged from the hospital, it would be a comfortable feeling for the physicians who have cared for the patient to be able to say, "That's that; now we can worry about the next case." In the majority of instances this point of view is correct, and possibly in the future it may be more frequent, but at the present writing this is not strictly so.

There are three sequelae following brain abscess which will be spoken of. The first is that of personality changes; the second, convulsions; and the third, recurrence of the abscess.

This young man first came under our observation four and one-half years ago. He was then age 7 years. Eight weeks prior to his admission to the Mount Sinai Hospital he had had a bilateral mastoidectomy performed, from which he made a satisfactory recovery, being discharged at the end of three weeks with granulating wounds. For 10 days at home he did well, but then began to have fever. A week later he complained of headache and shortly thereafter he began to vomit. Four days before he entered the hospital he had twitchings of the right side. These were soon followed by a generalized convulsion with loss of consciousness. When consciousness returned, his parents noted that the boy had some difficulty in speaking.

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On admission to the hospital the boy was semistuporous. His temperature was 99.6°, and pulse 80. The right mastoid wound was healed, the left was draining foul pus. A right hemiparesis was present. The deep reflexes were equal in the upper extremity. The right knee jerk was more active than the left. A positive Babinski reflex was obtained on both sides. A right homonymous hemianopsia could be charted and an anomia was present.

The spinal fluid was under a pressure of 90 mm. of water and contained 300 cells, all lymphocytes.

There was no way of being certain of the age of the left temporal lobe abscess, but it was thought to be upward of three weeks. The relatively small number of cells, all lymphocytes, in the spinal fluid tended to substantiate this.

Accordingly, two days after admission the left temporal lobe was explored. The usual resistance of a thick-walled abscess was not encountered, and thin brown fluid escaped from the lumen of the needle. Instead of drainage, the effect of simple emptying of the cavity and a small decompression was tried. Hemolytic streptococcus grew from the pus.

The operative wound had to be opened because of infection. Under observation, papilloedema began and advanced slowly, and on the ninth day after the aspiration the boy went into stupor.

At the second operation the brain was found bulging into the defect. The cortex was incised and retractors introduced into the cavity, which could be thoroughly inspected. It was drained with two tubes and a packing. This latter was removed on the second day. The tubes were gradually shortened. The final tube was removed permanently seven weeks after the operation.

Except for the anomia all the neurological signs had cleared up by the time the boy left the hospital. This latter gradually improved, although even today he will occasionally hesitate for a word.

In going into the history, we found that prior to his illness the patient, who was an only child, had been guarded too carefully and had not had little playmates. We thought then the condition of behavior would be temporary, but as time

has gone on the problem of management has increased and the character of the boy is changed, according to his parents, from what it was before the operation.

Mental changes we have seen with tumors, not only in the frontal lobes but elsewhere, above and even below the tentorium; however, following a removal of the tumor there has been a return to a normal state.

One cannot definitely ascribe the mental picture to the temporal lobe abscess. Neither can one definitely rule out the abscess as a cause, and I present the boy as showing one of the possible late complications.

The next two patients illustrate another complication.

Ten years ago, at the age of 16 years, this young man was admitted to Dr. Kramer's service with an acute left frontal sinus infection. He had been ill several weeks prior to admission and had had several chills, with rises of temperature to 104°. Aside from a nasal discharge and tenderness over the left frontal sinus, he presented a non-tender, fluctuant swelling in the hairline, and his optic discs were suggestive of early blurring. A radical external operation was done by Dr. Kramer. The frontal sinus was found filled with pus. A rough area of bone on the posterior wall was removed, exposing normal dura. During the next 10 days he grew more drowsy, alternating with periods of restlessness. His pulse dropped until it reached 48, and the eye ground picture advanced.

At this stage we explored. The swelling in the hairline was incised and pus encountered in the scalp, coming from a small opening in the bone. The red dura had granulations on it and through a pinpoint opening pus escaped. This was enlarged, a tube introduced and a brain abscess drained. Because, from the inadequate exposure, we could not tell whether we had placed the tube into a subdural collection or brain abscess, we had lipiodol X-rays, which showed the abscess to be in the frontal lobe. He was finally discharged, well.

Five years later he returned to us because of convulsive seizures, from which he had been suffering for four weeks. There were no neurological signs. Two encephalograms showed a normal left ventricle, but the right did not fill. We

never explained that to our satisfaction. He was discharged, but returned a month later, at which time a ventriculogram was done, showing normal ventricles on both sides. It was then decided to excise the scar tissue in the left frontal lobe. At operation done in October, 1934, the dura at the operative site and the core of scar tissue of the drainage tract was excised.

For two years he was free from convulsions, but in 1937 he was readmitted to the hospital following a convulsion. In January, 1938, he had a petit mal seizure, but since that time has had neither grand nor petit mal attacks.

The second patient who illustrates the development of convulsive seizures came under observation in 1929, at the age of 14 years. She was admitted two weeks after a tonsillectomy because of pain in the shoulder, chills and fever. On the Surgical Service she was operated upon for an osteomyelitis of the clavicle and, subsequently, of the femur. Her blood cultures were negative. Three weeks after her admission she had a convulsion which involved chiefly the left side, following which she had a mild weakness of the left face and arm. Five days later there occurred another left-sided convulsion. A month after the first convulsion she developed a right peripheral facial palsy, coincident with a right otitis media.

A myringotomy was done. Meanwhile, she showed signs of advancing papilloedema and frequent twitching of the fingers of the left hand.

Seven weeks after the first convulsion, a right frontal lobe abscess, situated in the Rolandic area, was drained by a tube. It contained about 30 cc. of thick, foul-smelling pus, from which an anerobic Gram negative bacillus was cultured.

Nearly six years passed before the patient began to have convulsive seizures. These were focal in character, involving the left face and arm. On one occasion the fit was purely sensory in nature. She was readmitted to the hospital, and examination showed slight weakness of the left upper extremity and impairment of two-point discrimination in that member. Airograms demonstrated a slight but definite retraction of the ventricles to the right.

The operative site was exposed — stimulation promptly induced a convulsion. The cerebral scar was excised. The

immediate postoperative result was a complete left motor and sensory loss, from which there was a gradual recovery.

Following this operation there was freedom from attacks for a year. They then returned about as before. In September, 1937, the operative site was re-explored. In addition to some scar, considerable yellow, soft brain tissue was encountered. The whole mass, about $2\frac{1}{2}$x$3\frac{1}{2}$ cm., was excised. Following this procedure, there was an increase in the paresis and sensory disturbance on the left side. The convulsive seizures continued, however, in a milder manner. The last known attack was in March, 1938.

In these two patients the relatively long free interval, over five years, between the drainage of the abscess and the onset of fits is to be noted. My impression is that, barring a recurrence of the tumor, I have not encountered such a long free interval following the removal of a brain tumor.

It should be pointed out that the excision of such scars following a brain abscess is not without some danger. I know of one instance, and others have reported instances, where organisms dormant in the scar have caused a meningitis years later when the scar was excised. In these days of chemotherapy, this could probably be prevented by the prophylactic use of sulfanilamide or sulfapyridine.

The fourth patient I wish to show illustrates still another possible complication.

In October, 1934, this married woman, age 39 years, was transferred to Mount Sinai Hospital from another institution. The following history was obtained and was substantiated by reports received from the hospital in Cleveland: Nine years prior to her present illness she had an earache on the right, and spontaneous discharge of pus from that ear. She developed headache, instability on walking and mental confusion. Five weeks after the onset a right cerebellar abscess was drained. A staphylococcus aureus was cultured from the pus. The patient remained well for three years. Then there was a return of headache, vomiting and drowsiness. She showed ataxia of the right arm and leg, early choking of the discs and tenderness at the operative site. An abscess in the right cerebellar lobe was drained a second time. This time a staphylococcus albus grew from the pus. When,

after an interval of six years, similar symptoms developed, the patient herself diagnosed a recurrence of the abscess. After suffering for three weeks she lapsed into semistupor and was taken to a hospital. Following a lumbar puncture, which yielded cloudy fluid with numerous polynuclear cells, she improved and was transferred to us.

Her temperature on admission was 103°, her pulse rate 98. The operative site was bulging and tender. The right ear was normal. The left showed evidence of a low grade acute inflammation (Dr. Maybaum). The neck was held rigidly and there was a bilateral Kernig sign. The deep reflexes were all hyperactive, the abdominals were absent and a positive Babinski reflex was obtained on both sides. A few nystagmoid jerks to the right were noted. The fundi were pale but flat. There was no ataxia.

The lumbar puncture yielded cloudy fluid at a pressure of 250 mm. of water and contained 2,600 cells, of which 82 per cent were polymorphonuclears.

Two days after admission the right cerebellar region was explored. The abscess was entered and 4 cc. of pus obtained at a depth of 5 cm. The exploring needle was dislodged and the abscess could not be located again. I had the feeling at the time that I had emptied the abscess. Culture of the pus grew out a staphylococcus albus.

Except for a superficial wound infection, the convalescence was uneventful, and she was discharged three weeks after the operation. She has remained well to date.

Had the right ear shown the signs of inflammation that were noted on the left side, we might suspect that the brain abscess on this last admission was a reinfection. But with a previous recurrence it is fair to assume this condition obtained again. The explanation must be that either a multilocular abscess was present from the start or that pocketing was allowed to take place following drainage.

These cases presented illustrate three possible late complications or sequelae, if you will, following operations on brain abscesses: *1*. Personality changes. *2*. Convulsive seizures. *3*. Recurrence of the abscess.

Can we do anything in our management of our patients to prevent these?

As to personality changes, I have no suggestions to make, nor am I sure of any method of preventing the convulsive seizures. I have seen them take place after simple tube drainage and after treatment which allowed herniation of the abscess. In general, that procedure which traumatizes least and leaves the least scar should lessen the chances of this complication.

The prevention of recurrences must depend on the thoroughness of the surgical care. Assuming that the original focus as a possible source of the infection has been removed, then the adequate treatment of the brain abscess should prevent a recurrence of that abscess. How that is best done I leave to Dr. King to tell you. Unless he disappoints, which he has never yet done, you will hear some sound surgical principles described.

4 East 95th Street.

SYMPOSIUM ON BRAIN ABSCESS.

(b) – MANAGEMENT OF BRAIN ABSCESS OF OTORHINOGENIC ORIGIN.*

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Brain abscess may result from five causes: *1.* Otitic infections — mastoiditis, otitis media, necrosis of the petrous pyramid, thrombosis of the lateral sinus, etc. *2.* Infections of the accessory nasal sinuses. *3.* Metastatic. *4.* Various diseases of the skull — osteomyelitis, malignancy, actinomycosis, etc. *5.* Traumatic. As the subject tonight is brain abscess of otorhinogenic origin, the other types will not be included in the discussion.

A brain abscess may be located in the cerebrum, cerebellum or brain stem, but only the first two will be covered tonight. Abscesses may be referred to as acute or chronic for the sake of discussion, but the surgical definition of a true abscess is that of a circumscribed and walled-off lesion. In the brain, the beginning of the process is a condition of localized encephalitis, regardless of the mode of entrance of the infecting organism. There is an inflammatory leukocytic infiltration with edema, the surrounding brain substance becomes softened, breaks down and necrosis ensues. The necrotic area liquefies and becomes a small core of pus and detritus contained in an ill-defined cavity without a real wall. About this suppurative focus there is continued necrosis of the brain substance surrounding the irregular, nonencapsulated cavity, with formation of more pus. The cavity is lined with shaggy necrotic tabs of hemorrhagic brain tissue, and beyond the necrotic zone there is a layer of markedly edematous brain substance.

If it is not limited, the suppurative process will extend into the meninges or ventricle, which may prove fatal. But should the protective reaction of the brain be sufficient, the accumulated pus is walled off or encapsulated, the edematous area

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is lessened, and a chronic brain abscess results — a most fortunate occurrence.

The shape of most abscesses is ovoid, with the smaller pole directed toward the infected bony cavity from which the infection has spread to form the abscess. Other abscesses, especially single metastic lesions, are more likely to be spherical. In instances where the abscess has spontaneously evacuated itself through the cribriform plate or the external auditory canal, the abscess may be in the shape of a fig, surrounded by pericapsular purulent necrotic brain substance.

The abscesses which are most amenable to surgical treatment range in size from that of a large malaga grape to that of an orange, but the size usually encountered is about that of a golf ball or tangerine. The average capacity is from one to three ounces in cerebral abscesses, and about one ounce or less in cerebellar.

The wall of an abscess varies from 1 mm. to one-fourth or one-half inch in thickness, the most commonly observed being about 2 to 4 mm. and requiring three to five weeks for its formation. There is also variation in the wall of the individual abscess, the thickness being greatest at the pole of the abscess near the involved bone. In other words, in temporal and frontal abscesses the wall is thickest at the external pole, and in most instances of cerebellar abscesses at the anterior or internal pole.

The formation of an abscess wall is influenced not only by the time of its duration but also by the type of organism producing the abscess, and the amount of resistance offered by the brain substance. In cases where certain anerobic organisms are present, there is little tendency to walling off, and the other extreme is represented by those cases in which the wall is firm, thick and resistant, due to the presence of a larger amount of fibrous tissue. The wall may even be so firm that the lesion is mistaken for a solid tumor — in fact, instances have been reported in which the entire abscess was removed as a tumor and its true identity was not suspected until the specimen was sectioned. But all are not alike in this respect, and some well defined abscess walls, even though 2 to 4 mm. thick, are soft, friable and easily perforated or torn.

In an early abscess that is not encapsulated, and before definite, firm encapsulation has taken place, the contents are not homogeneous. They consist of a supernatant yellow, oily, murky liquid and a residual thicker fluid made up of pus and brain debris or detritus. In chronic abscesses with a firm wall the pus is usually thick and yellow, or greenish-yellow. It may be odorless or it may be very fetid and foul-smelling. The amount varies from one or two teaspoonfuls to over seven ounces — the largest amount observed by the author in any case in which recovery took place.

Any of the pyogenic organisms may produce a brain abscess, the most common being streptococcus hemolyticus. Others are due to the pneumococcus, the staphylococcus, colon bacillus, etc., or the organisms may be mixed, while some abscesses contain sterile pus.

SOURCE OF INFECTION AND LOCATION OF ABSCESS.

Abscesses resulting from otitic infections are usually located in the homolateral temporal or cerebellar lobe, or both, the temporal region being more commonly involved. Involvement of the contralateral hemisphere may take place, in which case the infection spreads through the sinuses, but this is not common. The temporal portion of the temporosphenoidal lobe is involved more often than the sphenoidal. In the case of cerebellar abscesses, the outer inferior portion of the lobe is more frequently the site of the lesion, but it may be central in position.

An abscess secondary to infections of the nasal accessory sinuses usually involves the frontal lobe. As a rule it occupies the anterior pole of the lobe, extending backward, upward and outward, external to and above the anterior horn of the lateral ventricle; however, it may be situated in the lower internal portion of the lobe nearer to the falx and base than to the cranial vault. The homolateral lobe is usually the one involved, but the abscess may be in the opposite lobe, and the lesion is sometimes bilateral.

TEMPORAL LOBE ABSCESS.

Abscess occurs more frequently in the temporal lobe than in any other part of the brain. It is secondary, as a rule, to

otitic infections, and its presence may be recognized either before or after a mastoidectomy has been performed. Cholesteatoma, with necrosis of the dural plate and pathological exposure of the dura, may be found. The cortex of the mastoid may be hard and eburnated like ivory, while the bony areas near the dura may be soft and partly destroyed by the disease. The abscess may have existed for some time and a mastoidectomy may not have been performed.

The infection spreads from the diseased area in the middle ear along the following pathways:

a. Necrosis and perforation of the roof of the tympanic cavity. An extradural abscess may be present. The dura and other meninges become adherent to the necrotic bone area and to the brain, and the infection spreads directly into the brain substance, with the formation of an abscess. Most of these occur in patients between 20 and 40 years of age, for the reason that the roof of the tympanic cavity is much thinner and more friable in adults than in children. The external portion of the wall of these abscesses is situated very near the surface, may have a "stalk" connecting it with the diseased area in the skull, and may even perforate the wall spontaneously and discharge large quantities of pus through the external auditory canal.

b. Infection may extend or spread along the perivascular spaces of perforating blood vessels. In such instances the abscess wall is at a greater distance (1-3 cm.) from the meninges covering the temporal cortex. There may be no gross changes in the cortex.

c. Purulent thrombosis of the lateral sinus may be followed by abscess formation in the temporal lobe, but it is more frequently the cause of a cerebellar than a temporal abscess.

d. The infection may pass through the internal auditory meatus, but this seldom occurs. Petrositis involving the tip of the petrosa is more likely to produce meningitis than brain abscess.

Symptoms and Signs of Temporal Lobe Abscess: A common picture is that of the patient who has had a chronic, purulent discharge from the ear for a long time; or it may be

that a mastoidectomy has been performed and the patient afterwards does not do well. The wound does not heal completely or as well as it should. There is persistence of a foul-smelling, purulent discharge from the ear, or the discharge may have ceased rather suddenly.

The symptomatology and neurologic signs vary considerably according to the stage of development of the abscess. Pain in the ear, and later in the head, is one of the first symptoms. The pain is located in the temporal region, but radiates to the frontal and less frequently to the occipital region. It is not called a "headache" by the patient, but "pain." It is severe — sometimes excruciating — so that the patient may cry and moan. In children the crying is hard and prolonged, in contrast to the sudden short outburst of crying observed in well developed meningitis. The pain may be intermittent or continuous, and is due to the rapid increase of intracranial bulk resulting from the suppurative encephalitis, with its accompanying marked edema. Seldom does one fail to obtain the history of pain if the patient or family is carefully questioned. Loss of appetite and vomiting, not usually associated with nausea, occur early, and a mild or severe chill, or chilly sensation, lasting from a minute or two to a half hour, is experienced in most cases.

The pulse and temperature are elevated at first, more in children than in adults. There is marked prostration; the tongue is coated and there is a foul odor to the breath. During this time, in the first two or three days, the true diagnosis of the condition is seldom made, though it may be suspected. There is no choking of the discs or engorgement of the veins as yet, and high fever, stiff neck and the Kernig sign are present only when meningitis accompanies the formation of the abscess.

Should the suppurative area not be limited, the process usually extends into the ventricle, meninges, or both, and suppurative leptomeningitis ensues, followed by death. Should the process be limited and circumscribed by the establishment of a definite wall, the symptoms and signs change in character and degree. The pain is not so severe in many instances but may be marked in others. The patient may not complain so much of the pain because of the general diminished sensibility, diminution of the encephalitis and edema, or both. It

is believed to be due for the most part to the former, *i.e.*, diminished sensibility, for there is still marked increase of intracranial pressure and the hemisphere containing the abscess is always much larger than normal. The patient as a rule becomes lethargic, stares vacantly, and there is a loss of cerebration and attentiveness amounting to a dreamy state. One may gain his attention by sharply spoken words or commands, but he soon lapses again into an inattentive, listless condition.

The temperature ranges from normal to 102° F. Although some observers state that the temperature is normal, or even subnormal, most of the patients whom I have observed have registered a rectal temperature of between 100° and 102° F. No one has satisfactorily explained the low temperature. The pulse becomes slow — 45 or 50 per minute or less. It may be normal but it is not increased except in complicated cases. The respiration rate is also lowered but it is usually regular. There is rapid loss of weight, and on recovery the gain is likewise rapid.

Paresis of the face on the contralateral side is to be expected, but it never reaches the degree of paralysis seen on the homolateral side, due to the physiological or anatomical interruption of the facial nerve by disease or operation upon the mastoid. Only in large abscesses or in those with extensive edema does one observe paresis of the arm on the opposite side, and rarely is the lower extremity involved. Sudden hemiplegia of the opposite side is never seen except in instances of suppurative leptomeningitis or metastatic lesions.

Aphasia in some form is usually present in lesions of the left temporal lobe in right-handed patients and vice versa. The most frequent form is anomia — the use of the wrong word. The next most frequent type is circumlocution — the inability to name an object though the patient is able to state its use. There may be inability to write or spell. Motor aphasia is seldom observed, and when it is, one may know that the abscess is large and extends farther forward than usual.

Ophthalmologic examination usually gives significant data. The pupil may be dilated as a result of pressure on the IIIrd

nerve. The nerve is seldom completely paralyzed, and then only if meningitis is present. There may be paralysis of the VIth nerve with inability to look outward, the nerve involvement being due to highly increased intracranial pressure or to concomitant petrous tip involvement. The most constant finding is engorgement of the retinal veins, usually more marked on the homolateral side. Hemorrhages, fresh or old, may be seen, especially in abscesses which have existed for several weeks. Papilloedema is not observed early but may be marked — three to five diopters — by the time the patient comes to operation. The amount of swelling of the discs does not determine the size of the abscess. If the patient is co-operative, quadrantic or hemianoptic defects may be determined which are of value in localizing the lesion, and astereognosis is often demonstrated in co-operative patients.

The deep reflexes are usually increased, being greater on the opposite side. The Babinski sign on the contralateral side may or may not be present. The abdominal reflexes are usually diminished on the opposite side.

Lumbar puncture often reveals clear fluid under a pressure well over 200 mm. of water. The cell count may be normal but is usually increased to 20, 50, 100 or more per centimetre. Before encapsulation takes place, the cells are predominantly polymorphonuclear cells. When the cells are predominantly lymphocytes it is an indication that walling off of the abscess has been completed. Although rapid removal of a large quantity of cerebrospinal fluid may result in a fatality, it is not believed that the slow removal of about 2 cc. of fluid through a small needle for diagnosis is ever followed by untoward results. In fact, a careful lumbar puncture may at times be of therapeutic value.

The leukocytic and polymorphonuclear cell counts are increased as a rule, the former ranging between 11,000 and 18,000, with an average of about 14,000 cells per centimetre, and the latter from 85 to 92 per cent. Only in old, chronic, well walled-off abscesses is the count not increased. Radiographic films may be of some value, especially if a pathological exposure of the dura has occurred.

You have heard the words "usual" and "likely" repeated. From this you can readily infer that all cases do not always

present these signs and symptoms, especially when first seen by the neurologic surgeon. The patient may be in such profound stupor or coma that no history is obtainable from him, and very little definite information may be had from relatives or friends. On the other hand, the abscess may be so well walled off, benign and innocuous that no sign or symptom is present, and the fact that an abscess exists is established only at autopsy. The abscess may become calcified and give no further trouble. The contents may become sterile, the lesion quiescent and no trouble develop until the patient sustains a trauma, like a blow on the head. Then, the abscess may increase in size and rupture spontaneously into the meninges or ventricle with fatal termination. One of these complications usually arises if the abscess is not successfully operated upon but the patient may survive, ignorant of the presence of a brain lesion of any kind, and die of other causes. The abscess may rupture spontaneously through the ear but cure is not likely to be effected by this. Although death may result from greatly increased intracranial pressure which paralyzes the medullary centres, it is more likely to follow sudden rupture into the meninges or ventricle. In the former, death usually results in about six to eight days, and in the latter within 36 hours.

Diagnosis: One would be justified in concluding that a diagnosis should be made readily and easily after considering the points enumerated. In some instances this is so and the diagnosis can be made almost as readily as in the ordinary case of acute gangrenous appendicitis, but unfortunately it is not always the case. At times the diagnosis and localization of a brain abscess may be extremely difficult — sometimes even impossible.

When in doubt, one should resort to ventriculograms. Any case of temporal abscess which lends itself to operative interference will produce displacement and distortion of the lateral ventricles. Ventriculography is preferred to encephalography and is believed to be safer in these cases. Accurate localization of the abscess is most important if a successful outcome is to be achieved. Indiscriminate exploratory punctures with a cannula, or exploratory punctures through an infected field can be mentioned only to be strongly condemned.

Temporal lobe abscess should be differentiated from middle ear disease with adjacent edema of the brain, "serous

meningitis," extradural abscess, sinus thrombosis, brain tumor, spontaneous subcortical hematoma, chronic subdural hematoma and abscesses in other portions of the brain. Time will not permit consideration of these topics.

FRONTAL LOBE ABSCESS.

An abscess in the frontal lobe gives rise to the same general signs and symptoms as a well developed abscess in any other part of the cerebrum; namely, pain in the head, nausea and vomiting, foul breath, prostration, rapid loss of weight, slight deviation of temperature, slow pulse, fundus changes, leukocytosis, findings in the cerebrospinal fluid, changes in reflexes, slow cerebration, drowsiness, stupor and coma.

Localization and lateralization are usually much more difficult than in any other abscess. In fact, the neurologic findings may be very meagre, almost to the point of being nonexistent. When the general symptoms and signs of a brain abscess are present in a given case, the mere lack of localizing signs would make one suspect the frontal lobe as the probable site. This is especially true if the abscess is small or has reached a very chronic stage. The abscess may be very large, almost unbelievably so, and it is only when the abscess or the surrounding edema is extensive that lateralization may be determined. As the abscess or surrounding edema progresses backward, the two most helpful findings present are aphasia, when the "dominant" lobe is involved, and the paresis which the lesion produces. Paresis of the opposite upper extremity (weakened grip, etc.) occurs first, followed by that of the lower extremity, and last, if at all, of the face. In temporal lobe abscess the paresis is noticed first in the face, then the arm, and last, if at all, in the lower extremity. Flaccid hemiplegia has never been observed except where the abscess has ruptured into the meninges. Rupture into the ventricle will otherwise occur before the abscess produces complete hemiplegia.

Valuable aids in determining which lobe is involved are dilatation of the homolateral pupil and unilateral loss of smell. X-ray films of the sinuses, and the operative site, may also suggest the side involved, although the abscess may develop in the frontal pole opposite to the operative site or it may be bilateral.

In an instance where one has very definite reason for believing that an abscess is situated in a frontal lobe, exploratory puncture is advisable. If this is negative, ventricular puncture for ventriculography can be made through the burr hole. Where signs and symptoms are not of lateralizing value, ventriculography is preferred. The ventriculograms will show marked compression and displacement of the ventricle backward and downward.

CEREBELLAR ABSCESS.

Cerebellar abscess is more frequently found in children and younger people than in adults. The abscess is usually located in the lobe adjacent to the involved mastoid or lateral sinus, or both, is more frequently situated in the external portion of the lobe, and is smaller than the usual cerebral abscess. Frequently it is the shape of a bird's egg, with the smaller pole directed forward and firmly fixed to the posterior surface of the petrosa, from whence the infection extends; however, it may be located centrally within the cerebellar lobe.

From the foregoing, one can readily understand what the signs and symptoms might be. As the abscess increases the size and shape of the lobe which it occupies, pressure is made across the midline, resulting in displacement of the medulla and brain stem, and producing as a rule homolateral pyramidal tract signs, compression and blocking of the aqueduct, causing internal hydrocephalus. This results in marked increase of intracranial pressure, bilateral papilloedema, which may be equal, and bilateral frontal headache. Tenderness and pain on percussion may be elicited over that portion of the occipital region beneath which the abscess lies. There is frequently associated soreness and slight stiffness of the neck, more on the involved than on the opposite side. Nystagmus toward the affected side is usually observed.

Diagnostic lumbar puncture must be done with great care, and therapeutic lumbar punctures are banned. For relief of pain, ventricular puncture through a burr hole over the homolateral anterior horn is easily accomplished and should be done in every case. In most cases of cerebellar abscess observed by the author, the patients were stuporous or in coma. Following ventricular puncture the mental condition clears up almost immediately and the severe pains are

relieved. During this procedure air can be readily substituted for the cerebrospinal fluid, and ventriculograms should be made. These will show the degree of the dilatation of the ventricles. On the anteroposterior film the lateral ventricles will show moderate symmetrical dilatation, and the third ventricle will appear dilated and in the midline. On the posteroanterior view the posterior horn on the involved side will be smaller than that of the opposite side. This is due to the pressure exerted from below upward by the increased size of the involved cerebellar lobe, the pressure being transmitted despite the intervening tentorium. In labyrinthitis this does not hold true, and ventriculograms are, therefore, valuable in differentiating between these two conditions.

TREATMENT.

All of the extradural foci should be eradicated. Operations upon the mastoid and sinuses, without the use of mallet and gouge, should have been completed. Many "brain abscess pictures" will clear up after this, due to the fact that the condition was caused by edema, extradural abscess, etc. The treatment of the cerebral abscess is surgical but this does not imply that the operation must be done immediately in all cases. It is best done after the third or fourth week, at which time complete walling off and encapsulation of the abscess is expected. The increase in the cerebrospinal fluid cell count and changing of the majority of the cells from neutrophils to lymphocytes are indicative of better encapsulation. Should operative interference be deemed advisable, supportive dehydrating measures should be adopted. Blood transfusions may be indicated. Intravenous administration of 100 cc. of 50 per cent solution of sucrose is probably the best dehydrating agent. Its dehydrating effect is more prolonged than that of glucose, and its results are striking in some cases of abscess. It has been observed that a change from deep coma to that of clear mentality has followed administration of sucrose or glucose, and that the reverse was true when the solution was withheld.

When the proper time for operation on a cerebral abscess has been determined, the open method is preferred to the closed method of tube drains or tapping. In the open operation, advocated by the author in 1923 and used by him since

that time, the overlying bone, dura and cortex are removed, the bony opening measuring about 3 to 4 cm. in diameter. Most abscesses amenable to surgery are situated not more than 2 cm., and frequently much less, below the dura. This has been observed many times at operation and in museums. Through the dural opening the necrotic brain and pus outside the abscess cavity, should they be present, are removed, followed by the presenting portion of the abscess wall. The cavity is then readily evacuated and inspected, and secondary pockets or extensions are thus detected and dealt with. These pockets could not conceivably be drained by tubes, through a small drain, as in the closed method. Slow, controlled elevation of the floor of the abscess to the level of the skull accomplishes extinction of the abscess cavity, with prevention of secondary pockets, rupture into the ventricle and meningitis. Iodoform gauze is used in the cavity, and the area is well Dakinized until healed. The entire area is healed in from 35 to 60 days, and a scalp plastic with excision of the scar is done after six months.

Drainage tubes are used *only* in cerebellar abscesses, where there is no ventricle to be considered; where, in the majority of cases, the anterior pole of the abscess is fixed to the infected petrosa by adhesions which should not be separated, and where prolonged drainage of this area is proper; furthermore, no elevation or herniation of the floor of the abscess should be permitted on account of shifting or torsion of the brain stem and medulla. Preliminary ventricular puncture should always be done.

RESULTS.

Thirty-five patients have been operated upon according to the methods described, with four deaths: two from preventable pneumonia, one in a child, age 6 years, who was Dakinized and then placed by an open window in winter, the other in a comatose woman who had eggnog poured down her trachea; one in which the patient, in a distant hospital, removed the dressing and tore the brain substances with her fingers; and one in an unmanageable coast guardsman who got out of bed on the sixth postoperative day, resulting in rupture of the abscess into the ventricle. These four deaths were all temporal lobe abscesses.

In all cases, only one operation for brain abscess was required. Multiple procedures were never done. No patient has had postoperative convulsions except for four cases who had convulsions before operation.

As experience with these cases has increased and earlier observation of the brain abscess suspect has been made, the results have been better. The last 20 consecutive patients, with one exception, recovered.

140 East 54th Street.

SYMPOSIUM ON BRAIN ABSCESS.

(c) – BRAIN ABSCESS.*

DR. ABRAHAM KAPLAN, New York.

I deeply feel the great honor conferred upon me in addressing your Society. What I know about the diagnosis, treatment and complications of brain abscess I owe mainly to the teachings and guidance of Dr. Joseph E. J. King and Dr. Ira Cohen. From them I have learned to emphasize the following considerations in the study of patients with brain abscess:

1. Patients with brain abscess die as a result of one of any combination of three factors; namely, meningitis, spreading encephalitis and increased intracranial pressure. The pus *per se* within the cavity of a brain abscess is not responsible for the patient's death. Patients with well localized, uncomplicated brain abscess frequently fail to show signs of fever, leukocytosis or toxemia.

2. Further, from my experience with the patients I am about to present, I have been convinced that in the treatment of brain abscess the following four factors are of paramount importance: *1.* the initial and complete removal of the primary focus of infection; *2.* allowance of sufficient time for encapsulation of the brain abscess; *3.* accurate visualization of the size, shape and location of the abscess; and *4.* adequate and free drainage of the abscess after proper sealing off of the subarachnoid space to prevent meningitis.

Only the high points in each of the following cases will be given. The patients are presented in the order in which they came under my care.

Case 1: The first patient, an Italian shoe worker, age 39 years, seen in August, 1933, had a history of a chronic purulent discharge from the right ear since the age of 8 years.

For a month before admission he had headaches, nausea and vomiting. Upon admission the pulse was 60, temperature

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99° and there was some stiffness of the neck. Spinal fluid pressure was 380 mm., with 160 cells per cubic millimetre. A radical mastoidectomy was done. The following day he was drowsy, temperature 99-100°, pulse 50-85 and there was evidence of early papilloedema. In spite or because of the seriousness of his condition, he left the hospital against advice, only to return six days later in a coma, with a left hemiplegia, a left facial weakness and a stiff neck. Spinal fluid showed only 10 cells.

Operation under novocaine anesthesia was performed that night. A right temporal lobe abscess was drained with a Mosher basket (slide). The patient instantly regained consciousness on the operating table and began to move the left leg and arm. On the fourteenth postoperative day the drain was spontaneously extruded. The patient was discharged on the thirty-fifth postoperative day, symptom-free and with no abnormal findings.

Since Sept. 9, 1933, now over six years, he has been in excellent health, works 40 hours a week and states he never felt better.

This patient had the smoothest course of any brain abscess that has come under my care and probably can best be attributed to beginner's luck.

Case 2: The second patient, a housewife, age 32 years, was first seen in September, 1933. Since the age of 4 years she had had a chronic discharge from both ears, which led to lagging of speech. Three weeks before admission, discharge from both ears became profuse and the temperature rose to 104°. Soon she became drowsy, the neck was rigid but the fundi were normal.

A radical mastoidectomy was performed. Culture of the pus showed staphylococus aureus. Ten days later she showed right facial weakness, right hemiparesis and aphasia. Spinal fluid examination revealed 110 cells, 85 per cent lymphocytes. A left temporal lobe abscess was drained with a Mosher basket. On the sixth postoperative day she began to speak words, and thereafter motion in the right arm and leg began to return. She improved steadily and was discharged on the thirty-fifth postoperative day. The only residual symptom was a right homonymous hemianopsia (slide), for which she

learned to accommodate very quickly. She has remained well now over six years, able to do her housework and, according to her family, she has enjoyed better health during this period than for many years before.

Case 3: The next patient came under my care in May, 1933. Subjected to 11 major operations over a period of 17 months, he clearly illustrates the importance of complete removal of the primary focus of infection. In spite of repeated disappointments, his desire to live was so great and his courage so magnificent that one could not refuse him every possible chance. Though the physiological result leaves much to be desired, there is some satisfaction in having a live patient, for over six years now, with an acute and active mind and partially self-supporting.

In May, 1933, he presented the classical picture of acute empyema of the right frontal sinus, with fever, edema and tenderness over the right frontal bone, and ptosis of the right eyelid. Operation disclosed pus in the right frontal and ethmoid regions, as well as in the orbital space. Culture of the pus showed staphylococcus albus hemolyticus.

Six days after the operation he began to show weakness of the left arm and leg, early papilloedema, left facial weakness, stiffness of the neck and a bilateral Babinski. After the removal of the posterior wall of the frontal sinus an epidural abscess was found. The signs progressed to include a left hemianesthesia and left homonymous hemianopsia.

One month later he began to have left-sided Jacksonian fits. The papilloedema persisted, spinal fluid pressure was elevated but showed no cells. Ventriculograms were performed (slides). His condition was so critical that a right subtemporal decompression was performed as a palliative measure. For six weeks he showed surprising improvement and he was discharged.

Four weeks later he returned in coma, with generalized convulsions and temperature 104°. Spinal fluid showed 305 cells, predominantly polymorphonuclears, but no organisms. Within 10 days his condition improved. X-rays of the skull showed osteomyelitis of the right frontal bone. Using the previous incisions, a right frontal craniotomy was performed, with the removal of all the diseased bone (slides). The scalp was then sutured back in place.

A fortnight later the scalp was incised and the abscess drained with a Mosher basket (slides). The drain was removed at the end of four weeks. At the time of the discharge from the hospital he had no headaches, the fundi were flat, but he was left with a residual hemiparesis.

In February, 1934, two months after his previous discharge, he returned because of headaches. There was no fever or bradycardia. Aspiration of the previous site of the abscess yielded 120 cc. of pus. The abscess cavity was entered and the wall of the abscess marsupialized. A cerebral hernia developed. Aspiration in the temporal region yielded more pus. This aspiration was repeated and air injected, which showed that the hernia was part of the abscess cavity (slides). The cavity was opened and packed with gauze. On May 5, 1934, he was discharged, free of symptoms but with a residual hemiparesis.

He remained well until August, 1934, when a tonsillectomy was performed for recurrent sore throat. A week later he began to complain of headache. Aspiration at the previous site yielded 120 cc. of pus. Air was again injected, the abscess cavity reopened and treated by packing and Dakin irrigations as previously. Healing was prompt and he was discharged on Oct. 9, 1934.

For the past five years he has shown steady improvement. The skull defect has greatly filled in. He has no headaches. The fundi are normal. The residual hemiparesis has shown only slight improvement. He is now partially self-supporting.

Case 4: Two days following a fall in March, 1934, a school boy, age 15 years, began to have fever, headaches, stiffness of the neck and vomiting. Two weeks later he had diplopia, blurring vision and was unable to see objects in the left field of vision. Five weeks after the onset of the illness he began to have generalized convulsions.

When seen in May, 1934, there was evidence of great emaciation and loss of weight. He was drowsy, the neck was rigid, the fundi showed bilateral papilloedema, and he had a pronounced weakness of the left arm and leg, and astereognosis in the left hand. Lumbar puncture was under only 35 mm. of pressure with seven lymphocytes per cubic millimetre. Two diagnoses were considered — intracranial neoplasm and cerebral abscess.

A large flap was outlined on the right. Exploration through a single trephine over the right temporal lobe yielded pus. A Mosher drain was inserted. On the eighth postoperative day it was extruded spontaneously. Power in the left arm and leg improved. Three weeks after operation he was out of bed (slide), and on the thirty-third day after operation, July 7, 1934, he was discharged. Except for secondary optic atrophy, remaining left homonymous hemianopsia and left astereogenosis, there were no abnormal neurological signs (slide). He has remained well ever since and has learned to accommodate for the hemianopsia. He still has some astereognosis in the left hand.

Case 5: In April, 1934, following a mild cold, a school girl, age 12 years, began to complain of headaches and pain in the ears. In May, she had headaches and a convulsion with loss of consciousness. Lumbar puncture was negative. A week later frontal headaches returned, and within a fortnight there was evidence of a spontaneous discharge from the right ear. This was associated with fever, malaise, vomiting, transient diplopia and suspicious olfactory hallucinations.

She had tenderness over the right mastoid. The fundi showed bilateral papilloedema. On the left there was facial weakness, a mild hemiparesis and a Babinski sign. Spinal fluid showed a pressure of 240 mm. with 150 cells, 84 per cent lymphocytes.

While at the hospital she suddenly went into coma. At operation a right temporal lobe abscess was found, and culture of the pus showed streptococcus hemolyticus. For a month the postoperative course was not entirely satisfactory. Then, following a chill, the temperature rose to 103°, headaches and vomiting returned, and olfactory hallucinations were again noted. The left-sided paresis advanced. She complained bitterly of pains behind the eyes and down the right side of the nose, and soon she became drowsy. Aspiration of the wound yielded 30 cc. of pus, and 20 cc. of air was injected (slides). Films of the skull accurately show the size, shape and position of the abscess. A Mosher drain was inserted and within 48 hours all symptoms disappeared. The drain was removed a month later and she was discharged on Oct. 11, 1934. She has been perfectly well ever since.

Case 6: In November, 1936, this patient developed coryza and a sore throat. Three days later he had a chill, the temperature rose to 104° and was accompanied by pains over the left frontal region. He was treated conservatively for sinusitis. In spite of subjective improvement, he showed a septic type of fever, the temperature rising to 104-105°. Blood culture taken six days after the chill showed nonhemolytic streptococcus. A few days later another blood culture showed a similar growth. He was given prontosil intravenously and orally, and also a transfusion.

Seventeen days after the onset of the illness the patient had a Jacksonian convulsion involving the right side of the face, the right arm and leg, and he lost consciousness for one half hour. Prontosil was continued and he was given another transfusion. Four subsequent blood cultures showed no growth. Five weeks after the onset he became drowsy and behaved strangely. He had no fever but complained of severe headache, some diplopia, and vomited once.

When first seen at the hospital he appeared acutely ill and drowsy. There was stiffness of the neck, ptosis of the left eyelid, bilateral papilloedema, right facial weakness and weakness of the right grip. The spinal fluid showed 310 mm. of pressure and only 10 cells.

On Dec. 21, 1936, a trephine exploration of the left frontal area was performed, and a left frontal lobe abscess was found. A Mosher drain was inserted into the abscess cavity (slides). Culture of the pus showed staphylococcus albus. He improved promptly and by Jan. 19, 1937, 28 days after operation, the drain was removed. In March, 1937, he was discharged, symptom-free with no abnormal neurological findings.

Four months after his discharge, following an attack of acute tonsillitis with fever, he had several generalized convulsions with loss of consciousness.

In October, 1937, tonsillectomy was performed.

In March, 1938, in association with some fever, he had one generalized convulsion, and a month later he had several convulsions; however, since April, 1938, he has remained well in every respect and shows no abnormal findings.

73 East 90th Street.

THE USE OF COLLOIDAL THORIUM DIOXIDE IN ROENTGENOGRAPHY OF THE PARANASAL SINUSES.*

DR. RUSSELL C. GROVE, New York.

All rhinologists know how difficult it is to diagnose disease of the paranasal sinuses when there is no evidence clinically of their involvement. Especially is this true of those cases in whom a focus of infection is suspected. The sinuses transilluminate clearly and there is no pus nor polyps in the nose. Plain Roentgenograms may be reported negative or only slightly cloudy. In our experience in the allergy clinic of the Roosevelt Hospital we have found that further studies with certain of these so-called negative cases have shown that the sinuses were very definitely infected.

Formerly we injected these sinuses with iodized poppy seed oil, 40 per cent, and re-X-rayed them. We always suspected that in some of these cases the iodized oil, by causing such a dense shadow on the film, obliterated small polypoid changes or membranous thickening in certain portions of the antra; however, it was not until early in 1938 that we began to use colloidal thorium dioxide solution, and this was used only because we had a patient who was sensitive to iodine and we hesitated to inject iodized oil into his antrum. After consultation with Dr. Boone, of the X-ray Department, and Dr. Beisler, of the Urology Department, we selected umbrathor. We were very pleased with the films, and as there was no reaction we have used it subsequently in over 75 allergic cases. Solutions of thorium dioxide have been used for years in Roentgenography of the kidneys, bladder and intestine. More recently, it has been used in Roentgenography of the liver, spleen and the cardiovascular system by intravenous injection. The intravenous injection method has been criticized because of the possible toxic effects of the thorium, but injection of open cavities has been more generally accepted.

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Umbrathor is a colloidal solution containing 25 per cent thorium dioxide. It is a stable, brownish liquid, transparent in transilluminated light and of milky appearance in reflected light. After storage over a long period of time, a small amount of sediment may appear, which is not objectionable as it is resuspended upon shaking. It should be stored in a temperature of 60° or 70° F. No harmful effects from secondary rays have been ascertained. It is not absorbed and is nonirritating. No toxic reactions have been reported so far as we know, and we have observed none. It may be used in full strength or diluted with one to three parts of sterile distilled water. Too weak a dilution does not show up clearly, especially if the films are pale. It may be injected into the maxillary, frontal or sphenoidal sinuses through the usual trocar or cannula. We have not used the Proetz displacement method but I see no reason why it should not be satisfactory. We have irrigated most of the sinuses the next day to remove any remaining solution or residue. In some cases when this was not done we observed no ill effects, and when they were operated upon later a dry, brownish residue was sometimes found. We have X-rayed some of these cases daily for three or four days, and some several weeks or months later, and apparently it disappears rapidly if there is very little pathology or the membrane opposite the trocar has not been traumatized.

To summarize, we may say that: *1*. Thorium, being the second heaviest metal, is ideal for Roentgenography. *2*. That by flocculating on the surface, it is ideal for Roentgenography of organs which have rugae, such as the bladder and intestines; and, by comparison, polypoid degeneration of the paranasal sinus membranes. *3*. It may be used in cases sensitive to iodized oil. *4*. It is aqueous and may be injected easily in diluted strengths and is, therefore, inexpensive. *5*. It gives a clear, delicate delineation of slight polypoid changes which are often obscured by iodized oil. *6*. It is nonirritating and nontoxic.

We have injected over 75 antra, 30 per cent of whom have subsequently had a Caldwell-Luc operation, so that we have been able to verify the pathological changes.

I would like to show you a few of our Roentgenograms (demonstration of films).

60 East 58th Street.

CEREBROSPINAL RHINORRHEA PATHOLOGICAL FINDINGS.*†

DR. MAX L. SOM and DR. RUDOLPH KRAMER, New York.

The escape of cerebrospinal fluid from the nose may be the result of: *A.* Trauma. *B.* Neoplastic invasion of the skull. *C.* Infection. *D.* Increased intracranial pressure at times, called spontaneous rhinorrhea. *E.* Idiopathic.

A. Cerebrospinal fluid rhinorrhea consequent to trauma may be due to: *1.* An acute head injury or to a delayed complication of it. *2.* Postoperative leakage following cranial or rhinological surgery. In these instances the force of the trauma may produce a fracture of the base of the skull, with a tear of the dura and escape of cerebrospinal fluid. The dura is particularly adherent in the region of the cribriform plate, which is also the most fragile portion of the skull. At necropsy it is frequently possible to demonstrate the site of the fracture and the tear of the dura.

B. Neoplastic invasion of the skull and dura with fistula formation and resultant cerebrospinal fluid rhinorrhea is rare. The tumor of the skull may originate either in the bone proper, *viz.*, frontal osteoma, or may invade the bone by outward extension of an intracranial tumor, *viz.*, craniopharyngioma or pituitary adenoma. The fistulous tract from the cranial content to the nasal cavity is most often obvious, as in the case recently cited by Shea.[1]

C. Infection of the paranasal sinuses may produce a suppurative necrosis of the floor of the anterior fossa with an inflammatory perforation of the adherent dura. The resultant fistulous tract may then lead to escape of spinal fluid into the nose.

D. Cerebrospinal fluid rhinorrhea may be secondary to long-standing increased intracranial pressure associated with hydrocephalus or brain tumor. This has been included in a

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†From the Laryngological Service and the Laboratories of the Mount Sinai Hospital.

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group called "spontaneous cerebrospinal rhinorrhea" by numerous authors. Neoplasms of the pituitary or of the posterior fossa are more apt to produce internal hydrocephalus and are hence more often responsible for the rhinorrhea.

E. Idiopathic cerebrospinal rhinorrhea has been referred to as "pure spontaneous rhinorrhea" by St. Clair Thomson.[6] In such cases no disease was demonstrated and the patient was cured by rest in bed and frequent spinal puncture. The pathology of these idiopathic cases remains unknown.

Intranasal examination during the period of active rhinorrhea fails to definitely locate the site of the leakage even after the use of fluorescin (Friedberg and Galloway[2]).

The actual cause of the rhinorrhea in cases due to increased intracranial tension has not been established except in very rare instances. This ignorance exists, first, because of lack of postmortem examinations and, secondly, because complete gross autopsy generally failed to reveal any break in the dura or a pathway for the escape of the fluid in the nose. Pinpoint openings were occasionally recorded in the cribriform plate and dura (Ballon[3]). Some authors regarded these as congenital dehiscences (Livini[4]). Cairns[5] reported two cases of brain tumor with cerebrospinal fluid rhinorrhea in which he found a circular deficiency of the cribriform plate by Roentgen examination. Shea[1] cited a case with marked hydrocephalus and cerebrospinal fluid rhinorrea. At postmortem an erosion of the roof of the right ethmoidal labyrinth was found with a communication between the right anterior fossa and the nose through a fistula leading into an ethmodial cell. In this cell, hemorrhagic polypi were noted.

Sir St. Clair Thomson[6] had observed what he presumed was "an edematous fibroma" in the olfactory recess of a girl, age 25 years, with spontaneous cerebrospinal fluid rhinorrhea.

A perusal of all available literature fails to reveal any microscopic studies of fistulous tracts which start in the anterior fossa and communicate with the nasal cavity through a slit-like opening around which polypi are observed. In the routine study of serial sections of the paranasal sinuses at the Mount Sinai Hospital an opportunity of studying just such a fistula was recently afforded us.

Case E. F., female, age 44 years, who was known to have had a hypertension for many years, was admitted to the neurological service on Jan. 8, 1937. Five years previously, she had suffered an attack of excruciating headache, which was relieved only after lumbar puncture. For the past three years she has had intermittent, severe headaches, which were relieved by the escape of large amounts of clear, watery fluid through the nose, unaccompanied by sneezing.

On the day of admission she had a temperature of 102° and was totally irrational. There was a history of severe, unyielding headache during the past 24 hours. Examination revealed an acutely ill, obese woman, with evidence of an acute upper respiratory infection. Her blood pressure was 190 systolic, 120 diastolic. She was excited, disoriented and showed clinical signs of meningitis. There was a bilateral Kernig, a positive Brudzinski sign and a rigid neck. Lumbar puncture yielded a cloudy fluid with an initial pressure of 220 mm. of water. Cytologic study of the fluid showed the presence of 6,500 cells per cmm., 90 per cent of which were polymorphonuclear leukocytes. The patient was treated as a case of meningococcus meningitis with serum intravenously and intrathecally. There was noted a slight proptosis of her right eye. She received repeated spinal drainage. Bacteriological study of the fluid showed the presence of bacillus influenza. X-ray of the skull revealed a marked enlargement of the sella turcica with depression of the floor into the sphenoid sinus, a picture suggestive of some intrasellar tumor. Intranasal examination showed no evidence of sinusitis, but the rhinologist felt that the infection could have entered the cranial cavity through some defect in the floor. The patient gradually went downhill and died after a two weeks' stay in the hospital. A cranial autopsy was performed. A diffuse suppurative meningitis was found. A large pituitary neoplasm occupied the sella turcica. The paranasal sinuses were removed *en bloc*.

The sinus block was cut coronally in serial sections. In the right upper nasal cavity, between the attachment of the middle turbinate and the nasal septum, a polyp protrudes into the olfactory recess (see Fig. 1). Mesially, the polyp is separated from the septum by a gap which is at the mouth of a fistula connecting the nasal cavity with the anterior

cranial fossa. This tract starts endonasally in a defect of the olfactory mucosa and continues through the region of the cribriform plate until it reaches a dural gap just lateral to the crista galli. With higher magnification the tract is seen to be an inflammatory fistula (see Fig. 2). The dura is markedly thickened and infiltrated with polymorphonuclear leukocytes, which are especially abundant in the perineural sheaths. There is an acute osteomyelitis of the cribriform plate with bone erosion and sequestration.

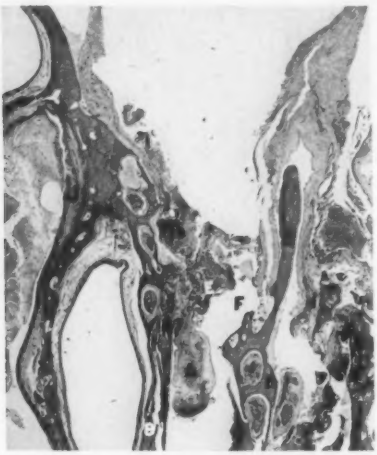

Fig. 1. Section through the cribriform plate showing dural polyp in the right olfactory sulcus. (A) Dural polyp. (B) Right middle turbinate. (C) Crista galli. (D) Nasal septum. (E) Cribriform plate with area of osteomyelitis. (F) Fistulous tract through which spinal fluid could escape.

Study of the polyp shows it to be covered by an extension of dura through what apparently was originally a normal cribriform perforation. The core of the polyp consists of nerve tissue which is enclosed by a prolongation of dura from the anterior fossa (see Fig. 3). Van Giesen stain demonstrates the continuity of the dura with the outer layer of the polyp. Actually, this polyp represents a herniation of the dura and nerve content through a cribriform perforation, probably as the result of prolonged increased intracranial tension. This dural polyp is lined superficially with a thin epithelial layer of nasal mucosa which it had pushed before

it. The dependent portion of the polyp is the site of an acute inflammation with ulceration and necrosis. This area probably represents the portal of entry of the meningitic infection. The inflammation rapidly spread to involve the dura and the bone of the cribriform plate and then invaded the meninges.

The fistulous tract in all likelihood represents the pathways of escape of the cerebrospinal fluid into the nose. One can conceive of the polyp acting as a plug and temporarily

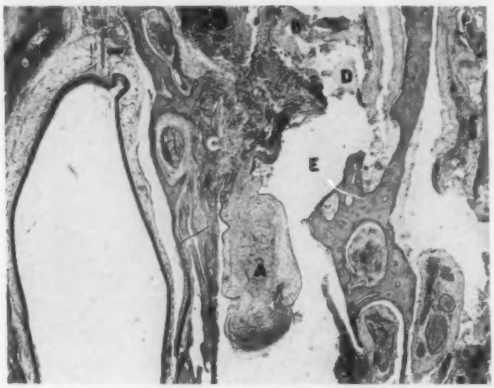

Fig. 2. Higher power of Fig. 1 at (A) showing dural polyp. (A) Polyp. (B) Inflamed dura over the cribriform plate. (C) Perineural infiltration. Note the continuity of the dura with the polyp. (D) Osteomyelitis. (E) Fistulous tract through which the spinal fluid could escape.

sealing the gap in the nasal mucosa and thus arresting the cerebrospinal fluid rhinorrhea. This might be accomplished by either change in position of the polyp or sudden increase in its size due to engorgement. At a later time the intracranial tension may be sufficiently high to cause a separation of the gap and start the flow of spinal fluid again. It is possible that the polypi noted by previous observers may have been of similar origin.

When infection of the base of the skull produces a cerebrospinal rhinorrhea, the patient sooner or later succumbs to a bacterial meningitis. In one such case, Thomson found a syphilitic necrosis of the floor of the anterior fossa at necropsy. Recently Wessely[7] reported a case of intermittent

spontaneous cerebrospinal fluid rhinorrhea in a child who died of meningitis, following an acute upper respiratory infection. At necropsy he demonstrated a defect about 0.5 cm. in diameter in the floor of the right anterior cranial fossa. There was a fistula leading into the right ethmoid with the herniation of the frontal lobe into the nasal cavity. We have observed a similar case recently.

Case C. N., male, age 50 years, was admitted to the Mount Sinai Hospital in August, 1939. One week following an acute

Fig. 3. Higher power through dural polyp. (A) Ulcerated and inflamed tip of polyp through which infection occurred. (B) Dural covering of polyp due to herniation of dura. (C) Continuation of nasal epithelium over the polyp.

upper respiratory infection, he suddenly became dioriented, complained of excruciating headache and vomited profusely. His temperature was 102.4° and he appeared acutely ill. There was some transient motor aphasia. On admission to the hospital, he had a constant cough with marked prostration. There was congestion at both bases of the lungs, with occasional rales but no dullness. There was no nuchal rigidity, and the patient was regarded as a probable bronchopneumonia with toxic encephalopathy. The following day his mental state became somewhat clouded, he had projectile

vomiting and developed meningeal signs. There was positive bilateral Kernig, positive Brudzinski and nuchal rigidity.

Lumbar puncture revealed cloudy fluid with an initial pressure of 220 mm. of water. Cytology showed 600 W.B.C. per cmm., 89 per cent of which were polymorphonuclear leukocytes. Intranasal examination showed evidences of an acute bilateral sphenoethmoiditis with early polypoid changes. Smear of the spinal fluid showed Gram positive cocci, which on culture proved to be pneumococci type XXIV. The patient

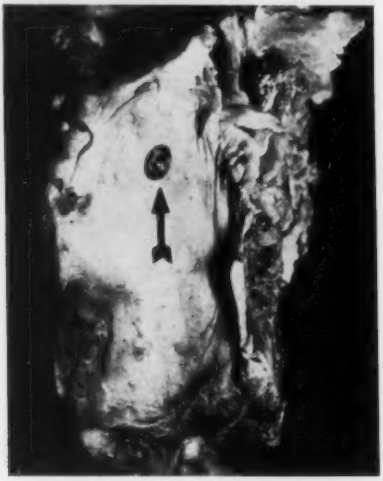

Fig. 4. Gross appearance of the posterior surface of the sphenoid sinus and clivus, showing macroscopic perforation. There was a free communication between the cavity of the right sphenoid and the subarachnoid space.

was immediately placed on adequate sulfapyridine therapy. He was given antipneumococcus type XXIV, rabbit serum and spinal drainage. Under local anesthesia, the left sphenoid and ethmoid sinuses were exenterated. The patient was stuporous and totally unresponsive. Polypi were encountered in the left sphenoid. Because the patient's condition was so nearly terminal, the right sphenoid and ethmoid were not entered. The following day it was noted that when the patient was placed on his left side there was an escape of a large amount of slightly blood-tinged serous fluid from his

right nostril. This fluid continued to flow freely and saturated several large pads. It became less blood-tinged and did not stiffen the linens. On turning the patient on his right side, the drainage from the nose ceased. Patient could rest only on his left side, otherwise the fluid trickled into his larynx and trachea and interfered with respiration. In spite of all chemotherapy the patient ceased, three days after operation.

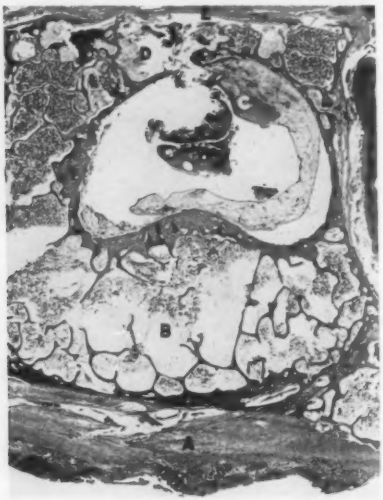

Fig. 5. Section through the sphenoid sinus showing a submucosal abscess with contiguous osteomyelitis. (A) Nasopharyngeal mucosa. (B) Floor of sphenoid. (C) Submucosal abscess. (D) Osteomyelitis. (E) Inflamed dura.

At postmortem examination a diffuse purulent meningitis was found. A defect in the dura about 1 cm. in diameter was noted on the cranial or posterior surface of the clivus (see Fig. 4). The parasanal sinuses were removed *en bloc*. The left ethmoid had been exenterated, and the left sphenoid was widely open. The right sphenoid was intact. A probe introduced into the natural orifice of the right sphenoid sinus readily passed into the cranial cavity through the defect previously noted in the posterior wall.

Study of serial sections revealed a submucosal abscess in the posterior wall of the right sphenoid sinus (see Fig. 5).

Adjacent to this abscess there is an acute osteomyelitis of the bony wall of the sphenoid sinus. The bone shows evidence of peripheral erosion and sequestration. The marrow spaces are infiltrated with polymorphonuclear leukocytes, and bacteria can be identified. The dura overlying the osteomyelitis area is inflamed. The sphenoidal mucosa containing the submucosal abscess is separated from the bony wall so that a free space remains. Sections taken more posteriorly show the osteomyelitis to be extensive and reaches up to the inflamed dura. At one point (see Fig. 6) the dura seems to

Fig. 6. Higher power of Fig. 5 through region of osteomyelitis. (A) Submucosal abscess. (B) Osteomyelitis with bone erosion. (C) Inflamed dura which has become thinned out at (D). (E) Irregular channel due to suppurative necrosis through which cerebrospinal fluid could escape.

be thinning out. A section more posteriorly shows the inflammatory defect in the dura (see Fig. 7). The dural edges are inflamed and infiltrated with polymorphonuclear leukocytes. A fistula can now be traced from the cranial cavity through the bone into the sphenoidal cavity. This represents the pathway of escape of the cerebrospinal fluid.

SUMMARY.

Cerebrospinal fluid rhinorrhea may be the result of trauma, neoplastic invasion of the skull, infection, increased intracranial pressure at times called spontaneous or idiopathic. Spontaneous cerebrospinal rhinorrhea is most frequently

associated with prolonged increased intracranial tension due to hydrocephalus or brain tumor. Although deficiencies in the floor of the anterior fossa were previously noted, the actual cause of the escape of the fluid had not been determined pathologically.

A case of pituitary neoplasm with spontaneous cerebrospinal fluid rhinorrhea is reported. Microscopic study of the sinus block reveals a fistulous tract leading from the floor of

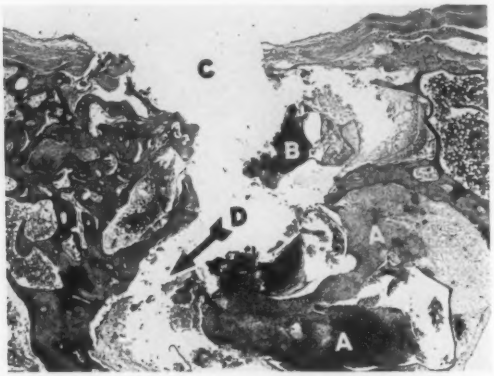

Fig. 7. Section posterior to Fig. 6 showing inflammatory defect of dura. (A) Submucosal abscess. (B) Osteomyelitis. (C) Defect in dura. (D) Irregular channel due to suppurative necrosis through which cerebrospinal fluid could escape.

the anterior fossa to the olfactory recess. At the mouth of the tract endonasally is a dural polyp which probably played a rôle in controlling the intermittent rhinorrhea and was the portal of entry of the bacterial meningitis.

Another instance of cerebrospinal fluid rhinorrhea due to an acute osteomyelitis of the posterior wall of the sphenoid is recorded. There is noted an erosion in the posterior bony wall of the sphenoid with an inflammatory defect of the overlying dura, thus creating a fistula for the escape of the spinal fluid. The portal of entry of the infection to the meninges is by way of a submucosal abscess.

The fatal meningitis in these cases almost always follow an acute upper respiratory infection. Patients with spontaneous cerebrospinal fluid rhinorrea should be guarded

against any upper respiratory infection and cautioned not to blow their noses, so as to avoid possible spread of the infection intracranially.

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121 East 60th Street.

TWO CASES OF ESOPHAGEAL FOREIGN BODY WITH COMPLICATIONS.*†

DR. FERNAND VISTREICH, New York.

The first case I wish to present is that of a woman, age 62 years. She was admitted to the bronchoscopic service of the New York Hospital with the complaint that four days prior to her admission she had swallowed a chicken bone, and that since then she had had increasingly severe pains in her throat and difficulty in swallowing. She had been seen by her local doctor, who said that he had removed the foreign body. Her general condition was good, and she had only a slight rise in temperature.

The X-ray picture showed a foreign body at the level of the seventh cervical vertebra, surrounded by a swelling of the esophageal wall. On close inspection, the area immediately surrounding the foreign body showed less density than the rest of this region.

Removal of the foreign body was undertaken the next morning. About one-half inch below the cricopharyngeal constrictor, the esophageal mucosa was red and edematous. Upon further progress of the esophageal speculum, thin flowing pus of slightly foul odor flooded the visual field. The estimated amount of pus was about three-quarters of an ounce, and it was removed by suction. Embedded in the posterior esophageal wall, in a pus pocket, a sharp-pointed chicken bone was found. The patient was kept in the hospital for four days and made an uneventful recovery.

This foreign body obviously penetrated the esophageal wall and set up a localized suppurative esophagitis, but very likely did not perforate the esophageal wall. Only this assumption would explain why the infection did not invade the posterior mediastinum.

An X-ray taken nine days later shows a normal outline of the esophageal shadow.

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*Read before the New York Academy of Medicine, Section on Otolaryngology, Dec. 20, 1939.

†From the Department of Otolaryngology of New York Hospital, Cornell Medical College.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 14, 1940.

The second case is that of a man, age 40 years, who was admitted to the New York Hospital with the complaint of a burning sensation on swallowing and substernal pains which radiated to both sides of his chest. The pains were especially severe a few seconds after swallowing. He believed that he had swallowed a piece of rib while eating lamb chops three days prior to his admission. His general condition was that of acute distress, with temperature 101° and pulse 120. X-ray examination failed to show a foreign body.

Case 2. Autopsy specimen showing perforating spicule of lamb bone as found in esophagus.

Esophagoscopic examination was undertaken soon after admission. There was an area of acute inflammatory reaction at a distance of 20 cm. from the upper incisors, but the foreign body was not found. The next day the patient continued in severe pain, unable to swallow fluids without adding greatly to his discomfort. The substernal pain diminished, and the temperature rose to 105° and the pulse to 150. The day following, the patient showed marked swelling of the cervical tissues but there was no subcutaneous emphysema. There was some dullness to percussion of the apices, which could be accounted for by the amount of swelling of the neck.

There were numerous rhonchi but no pneumonia rales. The temperature was around 104°, pulse 160, and the patient seemed extremely toxic and restless almost to the point of delirium.

The blood count at this time showed 20,400 white blood cells, with 72 adult polys, 16 immature polys, nine lymphocytes and three monocytes. Culture was negative. That same night the patient's condition became critical, the swelling of the neck increased and there was great difficulty in breathing — a fully developed picture of esophageal perforation with septic inflammation of the cellular tissue of the neck and mediastinum. The patient died at 1 A.M.

AUTOPSY REPORT.

About 2.5 to 3 cm. below the cricoid cartilage, a spicule of lamb bone, 2.25 cm. in length, with knife-like edges and sharp-pointed ends, lies nearly transversely across the lumen. Both pointed ends have perforated the esophageal wall. The esophageal wall immediately about the sites of perforation shows hemorrhage, suppuration and necrosis. The rest of the esophageal mucosa is well preserved and smooth throughout; however, the surrounding fatty and alveolar tissue, especially anterior and for a distance of about 8 to 10 cm., is indurated and granular. The cut surface exudes creamy pus, which in some areas forms small abscess-like pockets up to 1 cm. in diameter. There are numerous petechial hemorrhages in the adventitia of the aorta about the area of induration.

As can be clearly understood from this autopsy report, as well as from the specimen, the complication in this case arose virtually the moment the foreign body became lodged in the esophagus. Esophagitis, perforation of the esophagus and septic mediastinitis developed in rapid sequence; however, there can be no doubt that the case might have taken a much more favorable course if esophagoscopy had been done immediately, and not after a delay of three days. By the time the esophagoscopic examination was made, the local inflammatory reaction had reached such a degree that the foreign body became embedded in the folds of the edematous esophageal wall and could not be found. But even if the foreign body could have been removed, the prognosis would not have been substantially changed, because by that time the septic

mediastinitis must already have been in the first stages of development.

Although such complications are fortunately rare, according to the available statistics, the possibility of their development should be presumed in every case. Only immediately instituted expert management can reduce their occurrence to an absolute minimum.

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Meeting of April 12, 1940.

Erysipelas as a Complication Following Mastoidectomies. Dr. H. D. Harlowe.

Dr. H. D. Harlowe read his Inaugural Thesis, "Erysipelas as a Complication Following Mastoidectomies." Lantern slides were shown.

Sulfathiazol in Staphylococcus Infections of the Ear, Nose and Throat. Dr. Bert Levin.

Dr. Bert Levin gave a talk of "Sulfathiazol in Staphylococcus Infections of the Ear, Nose and Throat." Lantern slides were shown.

Sulfamethylthiazol in the Treatment of Staphylococcus Infections of the Eye. Dr. B. E. O'Reilley.

Dr. B. E. O'Reilley gave a talk on "Sulfamethylthiazol in the Treatment of Staphylococcus Infections of the Eye."

Hemolytic staphylococci have frequently been isolated from inflamed conjunctiva, but until recently have been considered as saprophytes by most workers. This assumption was based upon the failure to reproduce, by inoculation, the conjunctivitis in animals or in man. Recent work, however, has shown that an acute conjunctivitis in both animals and man can be produced by instillations of broth filtrates and also of staphylococcus toxin. This would lend support to the belief that staphylococci can and probably do produce a conjunctivitis by the liberation of an exotoxin; more important support for this view is accumulating daily in the reports of clinical improvement and cures from the use of specific immunologic procedures, such as staphylococcus toxoid, and more recently from the use of sulfamethylthiazol.

Incidence of Staphylococcus Conjunctivitis: Much of the work on this disease has been done at Iowa University by Thygeson, Allen and O'Brien; they place the incidence as high as 50 per cent of all ordinary catarrhal conjunctivitis; they base this upon a series of 2,110 consecutive cases of conjunctivitis in which complete studies of the bacterial flora were made; meholytic staphylococci were identified as the sole etiologic agent in 776 of the 2,110 cases, and also appeared in association with other bacteria in about 203 cases.

Diagnosis: Staphylococcus conjunctivitis is generally seen in the form of a chronic blepharoconjunctivitis, but is susceptible to acute exacerbations and remissions. Clinically, there is little to distinguish it from any other form of chronic conjunctivitis; as with any form of severe conjunctivitis, the lid margins usually appear red, scaly and somewhat thickened; there may be excoriations covered with crusts, and scales along the lid margins and in the region of the canthi; there may or may not be some injection of the bulbar conjunctiva; however, the following signs and symptoms are said to be more characteristic of staphylococcus conjunctivitis than other forms:

(1) The meibomian glands are usually infected and on expression yield a yellow mucopurulent material.

(2) The redness and thickening of the conjunctiva is always produced in the lower lid, especially in the fornix, caruncle and semilunar fold; in con-

trast, the conjunctiva of the upper lid and fornix shows only slight inflammation.

3. The discharge from the conjunctiva is strikingly scanty for the severity of the inflammation.

4. Severe cases may show an associated dermatitis in the lids and cheeks, characterized by small papular lesions, and in some cases even impetigo.

5. Practically every acute exacerbation is accompanied by a superficial punctate keratitis.

6. The disease is extremely resistant to the usual forms of treatment.

Treatment: In the past, treatment of this condition was very unsatisfactory and consisted of the usual procedures employed in any chronic conjunctivitis; i.e., the instillation of any and every conceivable type of astringent and antiseptic drops, and painting the conjunctiva with 1 or 2 per cent Agno3 solution. Recently the attempt has been made to immunize the patient to staphylococcus and its toxins by injections of increasing doses of staphylococcus toxoid. This is a staphylococcus toxin which has been detoxified by formalin and heat; it is obtainable in two dilutions and is injected intradermally or subcutaneously every fourth day in ascending doses. This form of treatment has met with some definite success.

(To be continued in a succeeding issue.)

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INDEX NUMBER

Vol. L—No. 12 DECEMBER, 1940

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Vol. L DECEMBER, 1940. No. 12

CONTENTS

ABSTRACTS FROM THE LITERATURE RELATING TO CONDITIONS IN THE TRACHEA AND BRONCHI. Dr. Charles J. Imperatori, New York - 1107

A REVIEW OF RECONSTRUCTIVE SURGERY OF THE FACE. Dr. Frank McDowell and Dr. James Barrett Brown, St. Louis - - - - - 1117

SYMPOSIUM ON BRAIN ABSCESS.

(a)—LATE COMPLICATIONS OF BRAIN ABSCESS. Dr. Ira Cohen, New York - - - - - - - - - - - - - - - - - 1139

(b)—MANAGEMENT OF BRAIN ABSCESS OF OTORHINOGENIC ORIGIN. Dr. Joseph E. J. King, New York - - - - - - - 1146

(c)—BRAIN ABSCESS. Dr. Abraham Kaplan, New York - - - 1159

THE USE OF COLLOIDAL THORIUM DIOXIDE IN ROENTGENOGRAPHY OF THE PARANASAL SINUSES. Dr. Russell C. Grove, New York - - 1165

CEREBROSPINAL RHINORRHEA PATHOLOGICAL FINDINGS. Dr. Max L. Som and Dr. Rudolph Kramer, New York - - - - - - - - 1167

TWO CASES OF ESOPHAGEAL FOREIGN BODY WITH COMPLICATIONS. Dr. Fernand Vistreich, New York - - - - - - - - - - - 1178

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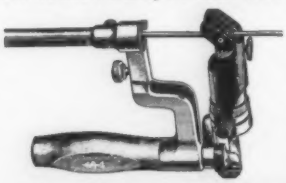

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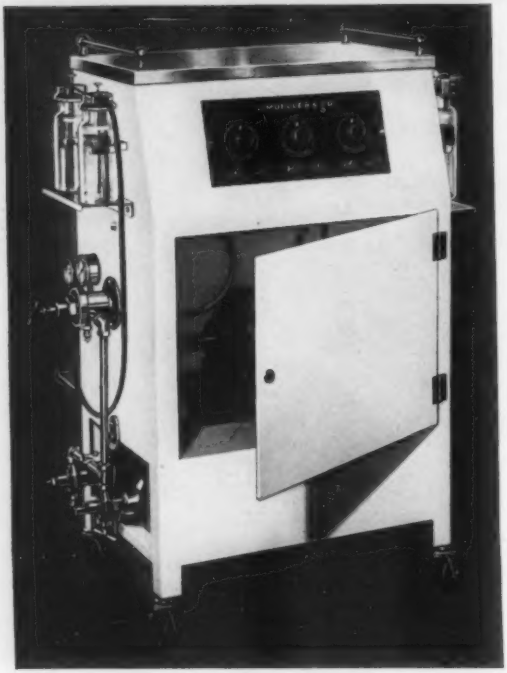

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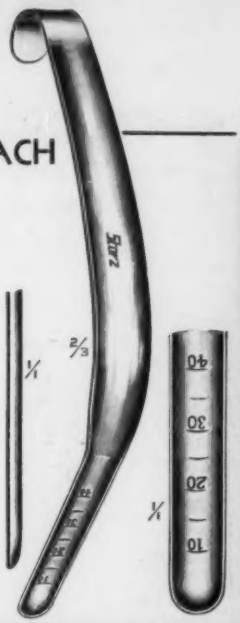

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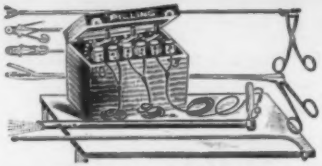

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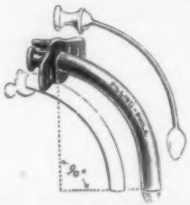

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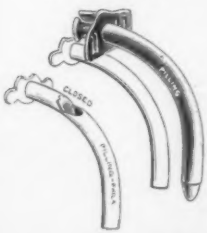

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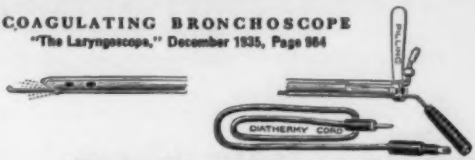
"The Laryngoscope," December 1935, Page 984



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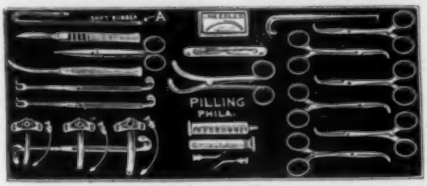

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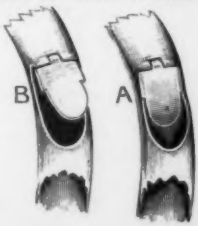

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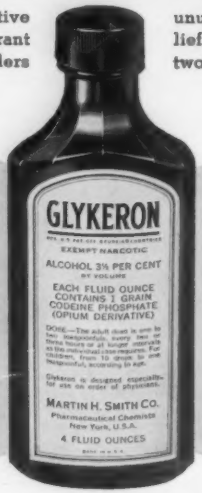

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